



UNIVERSITY OF DURHAM
PUBLICATIONS

THE PURPOSE AND PRACTICE OF MEDICINE

Selections from the writings of
SIR JAMES SPENCE

WITH A MEMOIR BY
SIR JOHN CHARLES

LONDON
OXFORD UNIVERSITY PRESS
NEW YORK TORONTO
1960

Oxford University Press, Amen House, London E.C.4

GLASGOW NEW YORK TORONTO MELBOURNE WELLINGTON

BOMBAY CALCUTTA MADRAS KARACHI KUALA LUMPUR

CAPE TOWN IBADAN NAIROBI ACCRA

© *University of Durham 1960*

*These selections were made by members of the Child Health
Department, King's College, Newcastle upon Tyne, Uni-
versity of Durham, and edited with the technical assistance*

of MR. P. B. DUREY, B.A., A.L.A.

PRINTED IN GREAT BRITAIN
AT THE UNIVERSITY PRESS, OXFORD
BY VIVIAN RIDLER
PRINTER TO THE UNIVERSITY

The majority of parents are stalwart people coping with life and remaining loyal to their responsibilities.

Family Studies in Preventive Paediatrics

The real work of a doctor is only faintly realized by many lay people. . . . This is a consultation and all else in the practice of medicine derives from it.

The Need for Understanding the Individual

An institution is the establishment and arrangement of human efforts towards a common end.

Institutional Medicine

The first aim of my department is comradeship, not achievement.

CONTENTS

MEMOIR. Sir John Charles

page 1

PART I

THE NATURE OF DISEASE

I. Cholaemia	27
II. The liver and pernicious Anaemia	39
III. A clinical study of nutritional Xerophthalmia and night-blindness	49
IV. Benign tuberculous infiltration of the lung (Epituberculosis)	51
V. The nature of disease in infancy	59
VI. Pink disease	71
VII. Poliomyelitis	80

PART II

THE STUDY OF DISEASE

VIII. Some observations on sugar tolerance	113
IX. Clinical tests of the antirachitic activity of Calciferol	130
X. Investigation into the health and nutrition of certain of the children of Newcastle upon Tyne between the ages of one and five years	142

PART III

CHILDREN AND FAMILIES

xI. The modern decline of breast feeding	<i>page</i> 161
xII. The purpose of the family	174
xIII. Family studies in preventive paediatrics	204
xIV. A thousand families in Newcastle upon Tyne	217

PART IV

THE CARE OF THE SICK CHILD

xv. The care of children in hospitals	233
xvi. Hospital beds for children	252

PART V

THE PRACTICE OF MEDICINE AND THE
TRAINING OF DOCTORS

xvII. Paediatrics in universities and teaching hospitals	263
xvIII. The need for understanding the individual	271
xix. Methodology of clinical science	281
xx. Institutional medicine	293

BIBLIOGRAPHY	298
--------------	-----

INDEX	303
-------	-----

LIST OF FIGURES, TABLES, PLATES, AND CHARTS

FIGURES

1. The average rise of red blood cells in 11 treated cases	<i>page</i> 43
2. Reticulocyte crisis and the rise of red cells during treatment	45
3. Age incidence of onset of pink disease	72
4. Monthly distribution of 404 cases of pink disease	73
5. Illnesses in a family of which one child had paralytic poliomyelitis	94
6. Blood-sugar curves of normal young adults	123
7. Comparing blood-sugar curves of infants with normal adult curve	124
8. Blood-sugar curves in old age	126
9. The stages of healing of rickets	133

TABLES

1. Admissions of cases of pernicious anaemia and their mortality	41
2. Erythrocyte counts in 20 cases of pernicious anaemia	42
3. Age distribution of poliomyelitis, England and Wales	85
4. Epidemiological pattern of poliomyelitis, Sweden, 1881-1911	91
5. The blood-sugar curve in infants under 12 months	124
6. The blood-sugar curve in children between the age of 2 years and 7 years	125
7. Blood-sugar curves in old age	126
8. Blood-sugar curves in cancer	127
9. The treatment and observation of human rickets	135
10. Zone of normal weight	147
11. Relation to the zone of normal weight of 3 groups of children	147
12. Relation to the zone of normal weight of 2 classes of children	148
13. Zone of normal height	148
14. Relation to the zone of normal height of 3 groups of children	149

15. Relation to the zone of normal height of 2 classes of children	<i>page</i> 149
16. Haemoglobin estimations for children from 'Professional families'	150
17. Haemoglobin estimations for children from 'Labouring and artisan families'	151
18. Social class by occupation of father	211
19. Place of birth of infants in the sample	211
20. Types of hospitals admitting 3,743 children	237
21. Ages of patients	238
22. Admissions in age-groups	238
23. Chief categories of disease	239
24. Population of Newcastle upon Tyne	255
25. Annual births and birth-rate	255
26. Annual admissions in age group 0-12 years	256
27. Annual admissions in age groups	256
28. Annual admissions to 'acute' hospitals for chief categories of disease	257

PLATES

1. Radiographs showing epituberculous infiltration of the lung. Case 1	<i>facing p.</i> 53
2. Radiographs showing epituberculous infiltration of the lung. Case 2	54
3. Radiographs showing epituberculous infiltration of the lung. Case 3	55
4. Radiographs from a child, aged 2 years, with rickets, treated with Calciferol	133

CHARTS

1. Weights of 124 children of 'Professional families class'	147
2. Weights of 125 children of 'Labouring and artisan families class'	147
3. Heights of 124 children of 'Professional families class'	148
4. Heights of 115 children of 'Labouring and artisan families class'	148

ACKNOWLEDGEMENTS

THE editor's thanks are due to the following for permission to reprint material which was first published elsewhere: The British Medical Association for 'Cholaemia: a clinical study of the nervous symptoms in liver atrophy' and 'Benign tuberculous infiltration of the lung' from *Archives of Disease in Childhood*; the Editorial Board of *The Newcastle Medical Journal* for 'The liver and pernicious anaemia'; the Department of Paediatrics, University Hospital, Uppsala, Sweden, for 'A clinical study of nutritional xerophthalmia and night-blindness' from *Acta Paediatrica*, vol. 11, 1930; the Editor of *The Lancet* for 'The nature of disease in infancy', 'Clinical tests of the antirachitic activity of Calciferol', 'The methodology of clinical science', 'Institutional medicine', and 'Hospital beds for children: an estimate of needs'; Messrs. Butterworths for 'Pink disease' from *British Encyclopaedia of Medical Practice*, 2nd edition, and 'Poliomyelitis' from *Modern Trends in Paediatrics* (ed. L. G. Parsons); *The Quarterly Journal of Medicine* for 'Some observations on sugar tolerance'; the City and County of Newcastle upon Tyne for 'Investigation into the health and nutrition of certain of the children of Newcastle upon Tyne between the ages of one and five years'; the Editor of *The British Medical Journal* for 'The modern decline of breast feeding' and 'The care of children in hospitals'; the National Children's Home for 'The purpose of the family' published by the Epworth Press; the Editor of *The New England Journal of Medicine* for 'Family studies in preventive paediatrics'; the Nuffield Foundation for the 'Introduction' and 'Conclusions and suggestions' from *A Thousand Families in Newcastle upon Tyne*; the Nuffield Provincial Hospitals Trust for 'Paediatrics in Universities and teaching hospitals'; the National Association for Mental Health for 'The need for understanding the individual—as part of the training and function of doctors and nurses'.

15. Relation to the zone of normal height of 2 classes of children	page 149
16. Haemoglobin estimations for children from 'Professional families'	150
17. Haemoglobin estimations for children from 'Labouring and artisan families'	151
18. Social class by occupation of father	211
19. Place of birth of infants in the sample	211
20. Types of hospitals admitting 3,743 children	237
21. Ages of patients	238
22. Admissions in age-groups	238
23. Chief categories of disease	239
24. Population of Newcastle upon Tyne	255
25. Annual births and birth-rate	255
26. Annual admissions in age group 0-12 years	256
27. Annual admissions in age groups	256
28. Annual admissions to 'acute' hospitals for chief categories of disease	257

PLATES

1. Radiographs showing epituberculous infiltration of the lung. Case 1	facing p. 53
2. Radiographs showing epituberculous infiltration of the lung. Case 2	54
3. Radiographs showing epituberculous infiltration of the lung. Case 3	55
4. Radiographs from a child, aged 2 years, with rickets, treated with Calciferol	133

CHARTS

1. Weights of 124 children of 'Professional families class'	147
2. Weights of 125 children of 'Labouring and artisan families class'	147
3. Heights of 124 children of 'Professional families class'	148
4. Heights of 115 children of 'Labouring and artisan families class'	148

MEMOIR

JAMES CALVERT SPENCE was born at Amble, Northumberland, on 19 March 1892, and died at Newcastle upon Tyne on the 26 May 1954. His life of sixty-two years spanned the last decade of the nineteenth century and the first half of the twentieth.

He shared with his coevals the swift and amazing transition from rich Victorian certitude to the feverish and restless present. He had known the warm sunshine of Edwardian schooldays, and had shared in the dangers and heroism of one world war, and worked in the shadow of a second. He had seen medicine change from a highly personalized profession, with the vestiges of a priesthood about it, into a rapidly moving symbiosis of art and science, with the science tending to outgrow the art.

For Spence and many of his contemporaries these sixty years have given the opportunity to live not one life but several, an experience with but few parallels in history. For him they provided also a field on which his special gifts and talents could be exercised to great advantage. It is appropriate that his achievements, the fruit of those talents, should be recorded.

Spence was born into a large Victorian household, being the seventh of a family of eight. His father, David Magnus Spence, an architect, and his mother, Isabella Turnbull, both of Northumbrian stock, were able to provide for their progeny a happy and comfortable home life. James, however, was wont to claim that despite a younger brother, very near to him in age, his childhood was a solitary one, and that he found his companions in books.

By the time he had become a senior medical student, the family were living in Teesdale at Lartington Hall. It was in this setting that Spence acquired that feeling for the countryside which whatever his circumstances or surroundings was always a consolation and a delight.

After his schooldays at Elmfield College, York, where he may have acquired 'small latin and less greek' but had learned to play a vigorous and intelligent game of soccer, he registered as a medical student at the University of Durham College of Medicine, Newcastle, in October 1909. From then onwards his career as an undergraduate was uneventful and uninterrupted by any of the common

academic casualties. He achieved neither prizes nor distinctions, until in June 1914, he qualified, to the undoubted surprise of his fellows, with second-class honours.

As a student he was casual and yet able, picking up knowledge quickly and retaining its essentials without difficulty or special application. He displayed considerable prowess on the football field, playing soccer both for the university and the medical school. But his best recollected extra-curricular activity was his membership of the Officers' Training Corps. Never a parade-ground soldier Spence enjoyed the summer camps and the Easter sojourns in barracks, mainly perhaps for the light-hearted camaraderie of canteens and their civilian equivalents, and for the opportunities they gave for talk, unrestrained and wide ranging, the talk which fashions students of all disciplines into civilized human beings. And that habit of talk, of whimsical speculation, and challenging argument remained with him always.

Of other activities there is little if any record. Spence was not particularly associated with the Students' Medical Society, nor did he ever join that select band, which over a collation of cake and cocoa gathered monthly in Professor Ranken Lyle's house to oversee the gestation of each number of the Gazette. This carefree period ended with qualification. Six weeks later the whole halcyon epoch of which it was part faded away with the outbreak of war on 4 August 1914.

Spence, after an attempt to join the Royal Navy which was thwarted by a minor degree of dental inadequacy, was commissioned in the Royal Army Medical Corps. He was posted to a Field Ambulance in a new army group of north-country battalions and supporting units, which ultimately became the 11th Division. With that Field Ambulance and Division, apart from a spell as Regimental Medical Officer to the 11th Manchesters, he remained throughout the war, serving at home, on Gallipoli, and in Egypt, France, and Belgium. After many months of preliminary training in this country, the Division found itself in the late July of 1915 on the island of Imbros ready poised for the ill-fated final Gallipoli offensive, based on the Suvla landing. On the morning of 7 August, the 11th Division, in the words of John Masefield: 'landed in the pitch darkness, by wading ashore, in five feet of water, under rifle fire, on to beaches prepared with land mines'.

Spence landed with his Field Ambulance, which was immediately

brought into action, amidst the surrounding congestion and confusion.

Masefield's vivid description of the components of that chaos continues as follows: 'ammunition boxes being passed along, water-carts and transports being started for the firing line, wounded coming down . . ., doctors trying to clear the way for field dressing stations, with every now and then a shell. . . .'

The attack failed, and a state of stalemate ensued. Then followed the months of heat, flies, dysentery, fatigues, and trench warfare. Through all this Spence continued unrelieved, suffering from a duodenal ulcer, and learning from personal experience the truth of the saying, sometimes in after days attributed to him, 'What is a little dysentery among friends.'

He remained until the evacuation on the moonlit night of 19-20 December. It has been fitly said: 'No man of that force passed down those trenches, the scene of so much misery and pain and joy and valour and devoted brotherhood without a feeling of sadness.'

For James Spence they were a lasting and triumphant memory. But it was in France and Belgium during the remaining two and a half years of the war that he acquired the reputation of being an exceedingly gallant and resolute bearer officer during offensives. Later he established himself as an able, experienced, and understanding Second-in-Command of his Field Ambulance.

Life was a succession of interchanging periods of action and inaction, of intense preparation for those offensive operations in which bearer officers were expendable participants, of rest in billets, and of infrequent leave. Spence was awarded the Military Cross in September 1917, and a bar thereto in the following year. The citation for the earlier award recorded 'his conspicuous gallantry and devotion to duty in proceeding to tend the wounded under heavy fire'; the later one commended his 'untiring energy and complete disregard of danger'. Nevertheless his medical interests did not fail him, because on one occasion the brief notes on a casualty sent down the line attracted the attention and obtained the outspoken approval of one of two consultant physicians visiting a casualty clearing station. The other was prompt to remark, 'Ah well he was one of my clerks.'

Shortly before the end of the war Spence was able to spend a leave at Lartington, and to go fishing with an old fellow student and comrade in arms on Gallipoli, Dr., now Sir Samuel, Bedson.

It was to Lartington also that he came on demobilization and prior to his return to Newcastle to take up an appointment as House Physician to Dr. Thomas Beattie at the Royal Victoria Infirmary. Dr. Beattie was meticulous in his use of military titles, and 'Major Spence' was frequently on his lips. Six months later, still as it were *in statu pupillari*, Spence showed his paediatric leaning by becoming for six months Casualty Officer at Great Ormond Street. This was followed by an appointment which has been described as in 'the borderland between medicine and biochemistry', for such, broadly, was the content of the John and Temple Fellowship at St. Thomas's Hospital which he held for two years. His work lay in a field of which he had no previous experience, and its fruits are to be found in the papers on sugar tolerance and the degenerative diseases of the liver—the latter given at the annual meeting of the British Medical Association in Glasgow in 1922—with which he made his entrance into scientific medicine.

Early in the tenure of his fellowship James married Kathleen Downie Leslie of Aberdeen and they commenced their housekeeping in a flat in Roland Gardens, South Kensington. That partnership, ideal in every respect, brought in the years to come joy not only to its members, but to the hosts of friends who were conscious of its perfection and serenity. The period of residence in London provided Spence with a completely new range of contacts, many of whom were to remain his firm and constant friends. It ended by bringing him directly within the orbit of the Medical Research Council. The Annual Report of the Council for the year 1921-2 contains for the first time the name of J. C. Spence. The occasion of the connexion though abortive at the time was interesting and is thus recorded:

Special arrangements were made during the past summer and autumn for a study of this frequently fatal disease (Epidemic Infantile Diarrhoea) at the suggestion of members of the staff of the Great Ormond Street Hospital for Children. . . . Dr. Donald Paterson . . . was in charge on the clinical side, Dr. G. M. Findlay . . . was responsible for the bacteriological and serological work, and Dr. J. C. Spence was biochemist. . . . It so happens however that the disease was happily rare in the past cold and rainy summer and that only six true cases became available for observation.

In the summer of 1922 Spence's old teaching hospital, the Royal Victoria Infirmary, decided to appoint a Medical Registrar and

Chemical Pathologist to its staff. Spence applied and his growing reputation as a biochemist and his local associations helped to clinch his appointment, which he took up towards the end of the year. He filled a gap in the local pathological department, and at the same time was available in the wards to help in the introduction of those exciting developments in the treatment of diabetes which were beginning to come through from Canada. The trend of the work was undoubtedly towards adult medicine, but a counter-balancing paediatric interest was at hand.

In the later stages of the First World War Miss Greta Rowell had prevailed upon an old friend, Mr. Frederick Milburn, to buy a house in the west end of Newcastle which became one of the earliest day nurseries in the country. By 1923 the need for such accommodation was greatly diminished but the more urgent need 'for a place where babies suffering from feeding difficulties could be scientifically looked after' had become manifest. The Day Nursery underwent the transition into a Babies' Nursing Home or Mothercraft Training Centre, and with this work Dr. Spence's name was associated in the annual report. A year later, in 1924, he had become Honorary Consulting Physician to the nursery. In 1925 the nursery became the Babies' Hospital, and Lady Ridley has sympathetically described some of its early vicissitudes in her fascinating historical note:

Dr. Spence was beginning to cause the committee a good deal of financial anxiety. He was asking for a Mercury Vapour Lamp which would cost £40 to £60, and (so the minutes run) he would also like an X-ray apparatus as it is most useful in diagnosing disease quickly and would save a good deal of experimenting in foods.

Before very long Spence had gathered together a team which included William Wardill as surgeon, Gavin Muir as general practitioner, and Elizabeth Cummings, matron. For the interested medical visitor there were from an early date Wednesday noon-day lunches of sandwiches and coffee, much talk and discussion, and not a little retrospective clinical auditing. In the background also was Lady Ridley as Chairman of Committee. Within a few years their combined activities had brought the hospital a reputation far transcending the homely surroundings in which its work was carried out. It became a living testimony to the truth of the Thucydidean statement that it is the men (and the women) and not the walls or the ships that make the city.

The Babies' Hospital, where the mothers are admitted to nurse their own children, has been described as 'the essence of Spence's achievement; the first and maybe the greatest expression of his sensitive insight into the nature of family life'. The purpose is best given in his own words.

It is an advantage to the child. It is an advantage to the mother, for to have undergone this experience and to have felt that she has been responsible for her own child's recovery establishes a relationship with her child and confidence in herself which bodes well for the future. It is an advantage to the nurses, who learn much by contact with the best of these women, not only about the handling of a child but about life itself. It is an advantage to the other children in the ward, for whose care more nursing time is liberated. In teaching hospitals it is of further advantage to the students, who gain a practical experience of the form of nursing they will depend on in their practices and learn to recognise the anxieties and courage which bind the mothers to their children during illness: a lesson which fosters the courtesy on which the practice of medicine depends.

Spence in his march towards paediatrics was helped by another happy chance. The then Medical Officer of Health of Newcastle, Dr. Harold Kerr, had a decided and discriminating antipathy to the exclusive employment of whole-time medical officers in *maternity and child care*. He preferred wherever possible to enlist in these activities men and women who, filling other medical appointments, were sufficiently interested to give some portion of their time to the work of the Maternity and Child Welfare Clinics. (It so happened that in the efflux of time three of these part-time medical officers filled respectively the Newcastle chairs of medicine, obstetrics, and child health). Spence established his first contact with the City Health Department in 1924 by becoming responsible for the conduct of a child welfare clinic. It was an association which was to continue in one form or another for the next thirty years, an association not only of mutual benefit to the parties immediately concerned, but of incomparable significance for the development and future of social paediatrics in this country.

In 1926 he was awarded a Rockefeller Foundation Fellowship which enabled him to spend an academic year at Johns Hopkins Hospital, Baltimore. He was accompanied by his friend and colleague Arthur Frederick Bernard Shaw, the Lecturer in Pathology, who also had been awarded a Fellowship. For Spence this was an exciting adventure, and as a consequence of it he became a

convinced admirer and the outspoken defender of all that is best in the American way of life and, in particular, of its devotion to the scientific approach to medicine as manifest at Johns Hopkins.

Compared with James Spence, Shaw's mind was cast in a different mould. His background as befitted a member of that great clan of Bernard Shaws, whose lustre was not limited to the protean G. B. S., was culturally and philosophically comprehensive. His influence upon James was also profound and continuous. Shaw helped to consolidate the foundations of Spence's scientific knowledge and understanding; he introduced him to conceptions of education and of the meaning of a university which twenty years later were to bear abundant fruit. Spence was not for long simply the mirror of Shaw's mind. He became ultimately both the interpreter and evangelist of his ideas.

The American experience was an enjoyable one and Spence made many new friends not only in Baltimore, but also in New York and Boston. On his return to Newcastle he was able, as a result of these American contacts, to introduce to the Infirmary the new knowledge of the liver and pernicious anaemia, the discoveries of Minot and Murphy of Boston and their colleagues. He described them in a paper before the Newcastle and Northern Counties Medical Society in November 1927 as 'a beautiful example of clinical study and research'.

In January 1928 he was appointed an Honorary Assistant Physician to the Royal Victoria Infirmary, and became more deeply involved in the business of hospital and consultant practice. At the Infirmary he had a heavy load of out-patient and teaching responsibilities; at the Babies' Hospital he was involved not only in teaching and clinical care, but in its domestic management also. And in addition there was the need for him to make a living in consultant practice, not amongst children—never in the provinces a rewarding field financially—but in adult medicine.

Yet despite all these commitments Spence was able, particularly between 1930 and 1934, to make outstanding contributions to clinical science. His papers on xerophthalmia (1931), epituberculosis (1932), and the classical demonstration of the antirachitic activity of calciferol (1933) belong to this period. This last investigation, undertaken at the request of the Therapeutic Trials Committee of the Medical Research Council, was one of the earliest and most conclusive of a now long series of controlled trials. It

established Spence's reputation as a clear-eyed investigator in the difficult fields where clinical acumen and scientific precision have need to walk hand in hand.

In the midst of this busy, absorbing life there erupted a controversy which excited his interest and sympathy, and brought him for the first time into the machinery of university government. A sudden and, as it ultimately transpired, an unhappy decision of the Council of the College of Medicine to dismiss Professor Harold John Hutchens, not from his chair, but from his Lecturership in Bacteriology, had been taken in March 1931. Thereafter ensued a succession of appeals to higher and higher authorities, until in the event a Royal Commission was set up under the chairmanship of Lord Moyne to consider the organization of the University of Durham. The outcome of the Royal Commission was the merging of the two Newcastle colleges into one, and simultaneously with the completion of that operation the appointment of Lord Eustace Percy as the first Rector of the new King's College. That event did not occur until 1937, but the six intervening years were not entirely wasted. Spence and many of his contemporaries learnt much from a close and at times intensive study of the minutiae of college and university government. They became experts in the administrative machinery of the Red Brick Universities, and Spence, in particular, studied the constitutions of all the English universities—ranging from Oxford to Sheffield. As a sign of his interest he contributed to the proceedings of the Royal Commission a paper, brief and cogent, upon 'The conditions of student life and work in the Newcastle College of Medicine'.

Running like a thread through all this varied activity was Spence's association with the Newcastle Health Department. In the 1932 report of the Newcastle Dispensary—an institution with nearly 200 years of charitable work behind it—there had appeared this pronouncement: '... the Committee are gravely concerned about the great increase in poverty, sickness and malnutrition amongst the poorest classes of the City.'

The Medical Officer of the Dispensary, Dr. Arthur Smith, had been even more positive, if lacking perhaps in scientific precision, when he declared that the blood of some of his adult female patients was 'half blood, half water'. The repercussions of the Dispensary report included a visit to Newcastle of the then Chief Medical Officer of the Ministry of Health, Sir George Newman,

and as a sequel of that visit, a request by the City Health Committee to Dr. Spence that he would carry out 'an investigation into the health and nutrition of certain of the children of Newcastle-upon-Tyne between the ages of one and five years'. Spence adopting the comparative method, contrasted city (or less euphemistically slum) children with those of the professional classes as to height, weight, the incidence of rickets, anaemia, and nutritional or deficiency diseases. His conclusions were clearly and explicitly stated—the main immediate causes of the apparent malnutrition of the 'city' children being the physical damage done by the infective diseases, promoted and perpetuated by indifferent housing and inadequate diet. This report which appeared in the early months of 1934 was the forerunner of the more elaborate and ambitious contributions to social medicine, culminating in the 1,000 families' study. It is a simple, straightforward document, and its importance lies not only in its intrinsic interest, but in its complete identification of Spence with the clamant problems of social paediatrics. He had become a medical sociologist.

From 1934 to the outbreak of war in 1939 Spence's life was composed of a series of well-established duties, never debased to the level of routine. They included the clinical supervision of the Babies' Hospital, his assignment at the Royal Victoria Infirmary as Assistant Physician to the firm which had Professor, now Sir William, Hume as its head, his work in the child welfare clinics of the Health Department, private consulting practice, and a self-imposed obligation to miss no opportunity for teaching. Such opportunities as were already provided in his hospital appointments were added to in the informal evening sessions with young keen minds in audience which were held at his home.

Spence in his middle forties was a stimulating and attractive figure, forthright and dogmatic and yet at times whimsical in utterance and wide-ranging in his interests. For eager and enthusiastic students he opened new vistas both in the clinical and sociological sciences, and laid before them also a miscellany of interests in literature, medical history, the physical arduours of fell-walking and rock-climbing. 'The countryside'—'the distant clearness in the hill'—always appealed to Spence. From the mid-twenties he always had some sort of rustic retreat—primitive indeed in the early years, but later enshrined in the seclusion of Grasmere and finally in the gracious surroundings of Middleton Tyas in lower

Teesdale. At all these places there was open house, and for many a student, house physician, or registrar they afforded opportunities for physical effort by day, and for the mental stimulation of free and flashing talk in the evening. The transition from the pentathlon to fireside polymathy was never too difficult. Most weekends found Spence against this country background and in this company, but his summer holidays were dedicated to Switzerland and the high Alps, and all the ineluctable mysteries which are the treasured possession of the mountaineer.

Another annual festival but of a different kind was the meeting of the British Paediatric Association at Windermere. This Association with its great significance for the future of paediatrics started in 1928 with Donald Paterson, Leonard Parsons, and James Spence foremost in its foundation. Here, in the ambience of lake and mountain, was that assemblage of friends which never failed to fire his enthusiasm. Professor Charles McNeil has drawn a vivid vignette of Spence at those meetings:

The British Paediatric Association was a very significant element in his professional life, and one which was the main source of his power and influence as a paediatrician. Its main business was the hearing and the discussion of scientific and clinical problems, but besides there was a good deal of policy-making. This is a sphere that is often looked at askance, or with lack of interest; but James Spence had all the gifts and talents that are required of the politician, and these he displayed at Windermere or other places of annual meeting.

In exposition his voice, though a little rough, had some of the sweet quality of tenor in it; and it was strong and bold because of the moral earnestness of his own mind. His hair was a little disorderly, but his clothes, strong thick tweeds—again gave the idea of rugged strength. All this was not only interesting but also pleasing and persuasive; yet something else in the way of character was needed, especially in a potential leader with many bold innovations to be tried. Spence had this extra gift; he belonged to the 'blessed' company of the pure in heart. On any 'political' issue, you would be sure to hear Spence either leading the discussion or making an important contribution to it: and he seemed to enjoy taking part. The effect upon his audience was very great; sometimes he stirred up opposition, and sometimes he secured hearty assent, but there was never any doubt about the purity of his motives. He showed no tricks of oratory; he made no perorations; there was always interest in his subject matter, which often sparkled with humour, and was always lit by the moral glow which possessed his mind.

One other event in the middle thirties worth recording was Spence's appointment as Paediatric Physician at the Newcastle General Hospital. The City Council had decided to build a complete new children's department at this hospital, and Spence, already the physician designate, was consulted about the plans. Before these were drawn he suggested that there should be a study of all the existing facilities for the care of sick children in Newcastle, and of the anticipated demand for hospital admission. Only when this had been done and an approximate total, appropriately divided into categories, had been arrived at did he turn his attention to consider the plans. He had already visited a number of continental departments, and had amassed a collection of relevant architectural literature. I can remember spending long summer afternoons with him in the minute garden of his house in Newcastle while the drawings of the new department were subjected to that painstaking, practically minded scrutiny which was the secret of Spence's success as an administrator. He was never the intuitively inspired creator of ideas. He preferred to be their painstaking and laborious alchemist.

Shortly after this Unit was opened in March 1939 it was visited by King George VI and Queen Elizabeth, and Spence had the opportunity to explain to his sovereign some of the achievements of modern medicine in improving the nurture of the premature infant.

In August of that year he decided before war came to have another glimpse of the Alps. He returned from his expedition to Switzerland by way of the Rhine Valley and in his car met with a number of adventures—not in circumventing obstructions or in encountering hostility, but because petrol like time was running out. To the old and experienced campaigner that he was the signs were ominous enough.

That same month he was appointed Honorary Physician to the Royal Victoria Infirmary, in the vacancy created by the retirement of his old chief Professor William Hume.

A little earlier in 1939 Spence had set on foot in association with his old student and later colleague, Dr. Fred J. W. Miller, a detailed inquiry into the deaths of such Newcastle children as might, within the period of a year, die before attaining their first birthday. The stimulus for this investigation was the infantile mortality rate of the city, which in 1937 had been recorded as 91 per 1,000 infants

born, a figure nearly 50 per cent. higher than the comparable rate for England and Wales.

It was a searching inquiry, involving much consultation with general practitioners and not infrequently modifications in the earlier diagnostic assessment. In the event 272 deaths which had occurred in 1939 were recorded, analysed, and discussed. Nothing epoch making was discovered but material had been so collected, collated, and interpreted that future action could be intelligently based upon the findings. No investigation so patiently conducted and diligently studied can fail to add something to the sum of knowledge. The report suggested that half the deaths were avoidable, and ended with the hope that the local infantile mortality might be reduced in the foreseeable future to 40. In the year preceding Spence's death—that is fourteen years later—that expectation had been more than realized, for the city infantile mortality rate was no more than 29.

The outbreak of war on 3 September 1939, while interfering to some extent with the progress of this inquiry, was the occasion of more immediate concern to Spence in other ways.

The Babies' Hospital, strategically sited at its foundation to provide for the needs of the population in the vicinity of the great Vickers Armstrong factories, became with the reactivation of all the old munition making undertakings exposed and vulnerable itself. Its evacuation to Blagdon, the home of the Riddleys, ten miles north of Newcastle, had been previously decided upon in agreement with the Ministry of Health. Lady Ridley has described some of the circumstances of that translation, and of the salient features of that adventure in its later stages. Her own words are the best record:

On the morning of September the 1st the cots and equipment were brought out to Blagdon and the nurses and babies followed in cars driven by members of the committee. By midday they were all installed but in a fearful state of muddle, when Mr. Cowell arrived with an acutely ill pyloric requiring immediate operation. I sterilised the instruments in a kettle on the kitchen fire and Mr. Cowell operated on the baby on a bath rack across the bath. The baby recovered.

We had no resident House Physician to start with (though later Dr. Mary Taylor visited the hospital daily in that capacity), and Dr. Spence, Dr. Elsie Wright, Dr. Ogilvie and Dr. George Davison took it in turns to come out every day on a weekly rota. Their untiring devotion to the

needs of the hospital was wonderful. The twenty mile journey to and from must have eaten into their time, but their willingness to come out at the shortest notice and at all times made it possible to carry on the acute nature of the work with the highest possible efficiency. On one occasion, when we had been snowed up for four days and were down to our last loaf of bread, and with a dying baby in the house, we managed in desperation to dig a way out for the car, taking four hours to reach Newcastle, where we found Dr. Spence putting on his gum boots and preparing to walk out.

At 7 p.m. on August the 22nd, 1944, a disastrous fire broke out at Blagdon. The hospital wing was never seriously threatened, nevertheless it was necessary to remove all the babies to outbuildings without delay. By great good fortune Dr. Spence happened to be doing a round at the time, and with the help of two R.A.F. men who had seen the smoke from the Great North Road and came to offer help, he organised the removal of the babies with complete calmness and resource. Later he returned to help fight the fire, and it was characteristic of him that he was one of the last people to leave the building, and indeed he was almost overcome by the smoke before he could be persuaded to do so.

The curiously unepisodic months of the autumn and winter of the first year of the war left many men and women, in the vivid phrase of Shane Leslie, 'rattling the loose change of time in their pockets'. They provided the opportunity for much discussion and talk on many subjects, and not a little speculation as to the future, particularly of medical education. In such fermentive discussions the didymi of Johns Hopkins Hospital, Shaw and Spence, were always prominent, and their first combined sally upon the traditional arrangements for professorial appointments in Newcastle occurred when the Chair of Medicine became vacant in 1940. Their desire to obtain the appointment of a whole-time professor was only partially successful, but their efforts paved the way for further developments. Spence himself had already been invited on several occasions to accept Chairs of Medicine in London and elsewhere but had always declined. It was therefore a surprise when the idea of a whole-time clinical professorship in Newcastle was brought to fruition by his appointment in 1942 to the newly created Nuffield Chair of Child Health. The University Appointments Committee and its assessors had no doubt as to their recommendation to the Senate.

In 1940 he became a member of that influential though unofficial forum, the Nuffield Provincial Hospitals Trust Medical

born, a figure nearly 50 per cent. higher than the comparable rate for England and Wales.

It was a searching inquiry, involving much consultation with general practitioners and not infrequently modifications in the earlier diagnostic assessment. In the event 272 deaths which had occurred in 1939 were recorded, analysed, and discussed. Nothing epoch making was discovered but material had been so collected, collated, and interpreted that future action could be intelligently based upon the findings. No investigation so patiently conducted and diligently studied can fail to add something to the sum of knowledge. The report suggested that half the deaths were avoidable, and ended with the hope that the local infantile mortality might be reduced in the foreseeable future to 40. In the year preceding Spence's death—that is fourteen years later—that expectation had been more than realized, for the city infantile mortality rate was no more than 29.

The outbreak of war on 3 September 1939, while interfering to some extent with the progress of this inquiry, was the occasion of more immediate concern to Spence in other ways.

The Babies' Hospital, strategically sited at its foundation to provide for the needs of the population in the vicinity of the great Vickers Armstrong factories, became with the reactivation of all the old munition making undertakings exposed and vulnerable itself. Its evacuation to Blagdon, the home of the Riddleys, ten miles north of Newcastle, had been previously decided upon in agreement with the Ministry of Health. Lady Ridley has described some of the circumstances of that translation, and of the salient features of that adventure in its later stages. Her own words are the best record:

On the morning of September the 1st the cots and equipment were brought out to Blagdon and the nurses and babies followed in cars driven by members of the committee. By midday they were all installed but in a fearful state of muddle, when Mr. Cowell arrived with an acutely ill pyloric requiring immediate operation. I sterilised the instruments in a kettle on the kitchen fire and Mr. Cowell operated on the baby on a bath rack across the bath. The baby recovered.

We had no resident House Physician to start with (though later Dr. Mary Taylor visited the hospital daily in that capacity), and Dr. Spence, Dr. Elsie Wright, Dr. Ogilvie and Dr. George Davison took it in turns to come out every day on a weekly rota. Their untiring devotion to the

needs of the hospital was wonderful. The twenty mile journey to and from must have eaten into their time, but their willingness to come out at the shortest notice and at all times made it possible to carry on the acute nature of the work with the highest possible efficiency. On one occasion, when we had been snowed up for four days and were down to our last loaf of bread, and with a dying baby in the house, we managed in desperation to dig a way out for the car, taking four hours to reach Newcastle, where we found Dr. Spence putting on his gum boots and preparing to walk out.

At 7 p.m. on August the 22nd, 1944, a disastrous fire broke out at Blagdon. The hospital wing was never seriously threatened, nevertheless it was necessary to remove all the babies to outbuildings without delay. By great good fortune Dr. Spence happened to be doing a round at the time, and with the help of two R.A.F. men who had seen the smoke from the Great North Road and came to offer help, he organised the removal of the babies with complete calmness and resource. Later he returned to help fight the fire, and it was characteristic of him that he was one of the last people to leave the building, and indeed he was almost overcome by the smoke before he could be persuaded to do so.

The curiously unepisodic months of the autumn and winter of the first year of the war left many men and women, in the vivid phrase of Shane Leslie, 'rattling the loose change of time in their pockets'. They provided the opportunity for much discussion and talk on many subjects, and not a little speculation as to the future, particularly of medical education. In such fermentive discussions the didymi of Johns Hopkins Hospital, Shaw and Spence, were always prominent, and their first combined sally upon the traditional arrangements for professorial appointments in Newcastle occurred when the Chair of Medicine became vacant in 1940. Their desire to obtain the appointment of a whole-time professor was only partially successful, but their efforts paved the way for further developments. Spence himself had already been invited on several occasions to accept Chairs of Medicine in London and elsewhere but had always declined. It was therefore a surprise when the idea of a whole-time clinical professorship in Newcastle was brought to fruition by his appointment in 1942 to the newly created Nuffield Chair of Child Health. The University Appointments Committee and its assessors had no doubt as to their recommendation to the Senate.

In 1940 he became a member of that influential though unofficial forum, the Nuffield Provincial Hospitals Trust Medical

Advisory Committee, and enjoyed the vigorous interchange of experience, which against an Oxford background, its discussions provoked. About the same time he became a member of the intensely active Committee on Medical Education set up by the Royal College of Physicians. He was also the first chairman of the College's Committee on Social and Preventive Medicine, which did so much to reorientate the teaching of Preventive Medicine, and to establish the status of social medicine in medical teaching. But further burdens were to be placed on his shoulders, burdens which because of their overwhelming interest for Spence were rather an excitement and a joy. In September 1944 he joined the Medical Research Council as one of its clinical members. He regarded this as perhaps the greatest of all the honours which befell him, to have the privilege for four years to sit in monthly session with minds richly endowed and scientifically conditioned, and in so doing to be abreast of all that was passing in the hastening panorama of medical science. His colleagues at the moment of his first appointment included Sir Henry Dale, his old friend Professor Samuel Bedson, Professor Leslie Witts, and Professor James Learmonth. Later Sir Alexander Fleming and Professor Harold Himsworth were of the company. Some months earlier he had become a member of another body, perhaps at first sight even more intellectually overpowering, the University Grants Committee. But he soon found that its atmosphere was easy and its fellowship congenial. When a few years later the Medical Committee was set up to deal with the specific requirements of post-war medical education his earlier experience of the broader problems which confronted universities at that time was an invaluable asset.

One of his colleagues on the University Grants Committee has written:

When he joined the committee in 1943 it had important work to do. The Government had realised from its war experience the unique importance of the universities to the country in its time of need. The Goodenough report was on its way, and a comprehensive health service was being mooted. When the war ended, the universities were faced with sudden and enormous expansion in staff, facilities, and students. Later the work of the medical faculties had to be coordinated with that of the National Health Service. This was the perfect milieu for Spence's genius. He brought to all those problems a vivid imagination and a warm humanity, and an unshakeable belief in the integrity of his fellow

men and their capacity to do the right thing, once they had realised what that was. He was impatient of the small mind, and the myopic vision; and he grew very restless when he saw that large and important views were being evaded because of tradition or detail. In the best sense he was at heart a revolutionary. He would repeatedly urge that a problem should be considered in its broadest aspects, and the decision based on principle and the future, rather than on expedience and the past.

Armed with this formidable experience Spence was an ideal witness for the purposes of the Goodenough Committee on Medical Education. Before that body, appointed in 1942 and reporting two years later, Spence appeared three times albeit in different roles. He represented both the University of Durham and the British Paediatric Association and appeared also *in propria persona*. Sir Ernest Rock Carling has remarked that the Goodenough Committee would not have been what it was without his sane and even humorous suggestions.

The Nuffield Chair of Child Health, though launched in August 1942, was not really under way until the end of the war, when it achieved its full establishment of staff. But from the beginning the Honorary Medical Staff of the Royal Victoria Infirmary had by a self-denying ordinance acquiesced in Spence's assumption of the control of the children's wards in the hospital. Even though the full complement of staff was not available, teaching of undergraduates was actively undertaken. Dr. Fred Miller, one of his earliest disciples, has given a simple and eloquent testimony to Spence's qualities as a teacher:

I first met him in 1932 while a third-year student, and like others was arrested and challenged by his personality, by his vividness, and above all by his combination of practical shrewd medicine with scholarship. He was not at this time popular with the majority of undergraduates, for he was often caustic, hating ignorance on one hand and 'smartness' on the other. Soon I found myself with a few companions at his house, becoming aware of the social needs of the community in which I lived, absorbing dreams of a life of study, friendship, and service.

He knew that students could not be taught paediatrics in three months, but he did think they could be sensitised to the needs of children and parents; that they could see parents and children treated with courtesy and understanding; that they could be shown an approach to the difficulties of helping sick children to build upon in later experience. For these things he regarded as the centre of the art of medicine, whether the student was later to work in family practice or in hospital.

There was much else to keep Spence occupied. The Babies' Hospital at Blagdon threw up its problems, sometimes of personnel, sometimes of elemental interference by snow or fire; the Medical Research Council and University Grants Committee were exigent in their demands, and in addition Spence became a member of the Interdepartmental Committee on the Care of Children, where he served under the chairmanship of Dame Myra Curtis, and visited not a few institutions still redolent of Dickensian atmosphere and Victorian practices. It was, moreover, during this period that he was invited to try his skill at one particular piece of institutional reorganization. Professor Donald Court has described his impact upon the recently translated Princess Mary Maternity Hospital:

Between 1944 and 1946, without a single alteration to the structure of the hospital, the mortality from infection in the newborn fell to a tenth the previous figure. Once he was clear that the essential principles of newborn care were understood by the doctors and nurses alike, and realising that a new hospital was a distant hope, he rallied the staff in the task of reasonable but limited reconstruction. Although the present visitor to the Princess Mary Maternity Hospital may see nothing more than the sensible adaptation of an unsuitable building, those who were close to James Spence through this period of renovation knew that for him it expressed an attitude of responsible stewardship and sensible economy which he sought for the health service as a whole.

By the beginning of 1946 things had fallen into shape in the new Children's Department at the Royal Victoria Infirmary and the team had been gathered together. That year saw the delivery at the Royal College of Physicians of the Charles West Lecture on 'The Care of Children in Hospital', a lecture which represented the fruit of his thinking over many years, and placed on the record his description of the fundamental change in hospital care, which arises when a mother comes into hospital with her child. Later in the year there came the detailed planning of the 'Survey of the 1,000 families'. That survey—the product of co-operation between the University Department of Child Health, and the City Health Department, then owing Dr. Stanley Walton as its chief—arose from the study of all the children born in Newcastle during May and June of that year. Diligently followed through until 1954, when a temporary reckoning was called and the first volume published, this survey provided one of the classical protocols and texts of social paediatrics in this country.

With the coming of 1948 and the impending inauguration of the National Health Service Spence found himself further committed willy-nilly in a number of ways. He was a member of the first Central Health Services Council, and of its Standing Medical Advisory Committee. These brought him in touch with the organization of the new service, while he preserved and maintained his previous detachment from its medico-political aspects. Throughout his career Spence although a member of the British Medical Association had channelled his interests into the scientific rather than the political endeavours of that body. Nearer home he was appointed to the Board of Governors of the Newcastle Group of Teaching Hospitals and there demonstrated that a whole-time professor of a clinical subject could be a valuable exponent of even the minute economics of hospital management.

To this highly complicated but yet integrated life of teacher, counsellor, and administrator, Spence was beginning to be called upon to add the exacting duties and responsibilities of a visiting lecturer overseas. The first of his major assignments in this role of academic ambassador was in 1948 as Inter-state Postgraduate Lecturer in Australia. It was a strenuous undertaking involving much travelling, but Spence revelled in it, and in the enthusiasm of those to whom he lectured and with whom he worked. Australian life in its spaciousness enraptured him. He found New Zealand rather different but equally hospitable and receptive. The following year he spent many weeks in the United States and Canada. He had been invited to give the Cutter Lecture in the Harvard School of Public Health, and his visit coincided with the eightieth anniversary celebration of the Boston Children's Hospital. It was my privilege to be present at the Cutter Lecture, which was a lucid review of the place of paediatrics in the life of the community, with some passing reference to the early experience of the 1,000-family investigation. The simple, conversational style, the humour, the north-country articulation and crispness of expression, the modesty and charm of the speaker fascinated the American audience of students and graduates. There were other occasions during that week in Boston for Spence to display his personal magnetism, his clinical acumen, and his ability to deal with any occasion whether planned or impromptu. Not even the arranged provocation of an American Clinico-Pathological conference could dismay him. It only served to display his versatility and prodigious memory. Following the

There was much else to keep Spence occupied. The Babies' Hospital at Blagdon threw up its problems, sometimes of personnel, sometimes of elemental interference by snow or fire; the Medical Research Council and University Grants Committee were exigent in their demands, and in addition Spence became a member of the Interdepartmental Committee on the Care of Children, where he served under the chairmanship of Dame Myra Curtis, and visited not a few institutions still redolent of Dickensian atmosphere and Victorian practices. It was, moreover, during this period that he was invited to try his skill at one particular piece of institutional reorganization. Professor Donald Court has described his impact upon the recently translated Princess Mary Maternity Hospital:

Between 1944 and 1946, without a single alteration to the structure of the hospital, the mortality from infection in the newborn fell to a tenth the previous figure. Once he was clear that the essential principles of newborn care were understood by the doctors and nurses alike, and realising that a new hospital was a distant hope, he rallied the staff in the task of reasonable but limited reconstruction. Although the present visitor to the Princess Mary Maternity Hospital may see nothing more than the sensible adaptation of an unsuitable building, those who were close to James Spence through this period of renovation knew that for him it expressed an attitude of responsible stewardship and sensible economy which he sought for the health service as a whole.

By the beginning of 1946 things had fallen into shape in the new Children's Department at the Royal Victoria Infirmary and the team had been gathered together. That year saw the delivery at the Royal College of Physicians of the Charles West Lecture on 'The Care of Children in Hospital', a lecture which represented the fruit of his thinking over many years, and placed on the record his description of the fundamental change in hospital care, which arises when a mother comes into hospital with her child. Later in the year there came the detailed planning of the 'Survey of the 1,000 families'. That survey—the product of co-operation between the University Department of Child Health, and the City Health Department, then owning Dr. Stanley Walton as its chief—arose from the study of all the children born in Newcastle during May and June of that year. Diligently followed through until 1954, when a temporary reckoning was called and the first volume published, this survey provided one of the classical protocols and texts of social paediatrics in this country.

own indomitable fortitude. Lady Ridley has written of those last weeks that

he was eager to know what other men had said of death, and dismissing Claudio's famous speech in 'Measure for Measure' as failing to agree with what he himself was feeling, he found delight in the detailed, almost clinical description of his father's dying in the first Lord Clarendon's autobiography. [And she continues] Even when he knew he was dying, he took a technical interest in the progress of his illness which was wholly objective and characteristic of his reserved stoicism. One of the last things he said was, 'To a biologist dying is a very ordinary part of life'. But James Spence accepted death with a courage that was not ordinary at all.

He attended his last meeting of the Medical Research Council on St. George's Day, 23 April 1954, and spent the night at his well-loved Athenaeum. At the meeting of the Council that afternoon he brooded and waited as he had so often done on previous occasions, until some of the tenuous cloud of discourse had been dispersed. Speaking for some ten minutes he gave a perfect epitome of his views on 'a problem' in Sir Harold Himsworth's words:

broader even than medicine itself. He was concerned with the whole question of the relationship of scientific knowledge to the intellectual development of our society, not in any vague or sentimental sense, but as it expressed itself in the concrete problem of integrating the effort of the scientific organisations of government with that of the universities.

He set out to show where stood the universities, and how they might suffer by the competition of organized scientific institutions of narrower outlook and more limited purpose.

It was a theme which he had developed a few months earlier when belatedly called upon to fill the Presidential Chair of the Newcastle upon Tyne and Northern Counties Medical Society and to deliver an address on 'Institutional Medicine'. It was also his last clear authentic message to any body official or corporate, for he died on the 26th of May.

This is no more than a biographical note, linking up the main dates, episodes, undertakings in a life varied, colourful, pervasive in its authority and influence, and withal complete. It does not purport to assess or to interpret the achievements of its subject. Nor is it an *oraison funèbre* or a psychological study, but rather a

plain record of tasks fittingly accomplished through which the merits and achievements of James Spence shine clear. It is inevitable that the inquiring minds of the future will ask the salient question:

Ah, did you once see Shelley plain,
And did he stop and speak to you
And did you speak to him again?

and having asked, it is right that they should receive a satisfying and an illuminating answer. Happily there is available amongst the many appreciations every kind of tribute and homage. These have been gathered together from the pages of *The Lancet* by one of Spence's oldest and greatest friends, Dr. T. F. Fox, the Editor of the journal. Here is a gleanings from that harvest.

Of the intimate nature of his influence Sir Harold Himsworth wrote:

The influence of James Spence was so largely personal that it is difficult to pay tribute to it so that future generations will understand the esteem in which he was held by his contemporaries. There are some men—the majority—who achieve eminence by building some part of the factual structure of knowledge. There are others—the architects of knowledge—who give shape to particular concepts. Both leave contributions which are evident, not only to their contemporaries, but to their successors. But there are yet others—a few—whose contributions lie in appreciating needs and creating the climate of opinion in which these can be translated into realisable concepts. Their influence is to be measured, not in terms of the factual contributions which survive them, but in the value that was placed on their advice during their lifetime.

Perhaps the greatest tribute that can be paid to Spence as a man is that his contemporaries were prepared to heed him and yet still held him in affection. If his character had been different, he might so easily have followed the commoner lot of the imaginative and become a mere rebel. He had, however, a fundamental generosity of temperament which conditioned his attitude to others and their response to him. This, combined with a very characteristic whimsical charm, endeared him even to those with whom he was in most disagreement. To juniors he was an inspiration. His curiosity was ever alive and he retained the capacity to see the fascination of the ordinary. But he never forgot the man behind the work; and there must be many who remember the appreciative aside which showed that the true measure of their achievement had not been overlooked.

Professor, now Sir George Pickering, another Novocastrian, described his charm in these words:

James Spence was perhaps the most attractive personality I have ever met. I never climbed with him but I have often thought that mountaineering expressed most clearly, perhaps, his personality. His gaze always seemed to seek the distant peaks and his mind delighted in the problem of how to achieve the difficult or seemingly impossible; nothing thrilled him so much as a new journey over difficult and often unknown ground. It was his zest for life and his capacity for regarding every phase and every incident in life as an exciting adventure that made him so enchanting a companion. This quality did not leave him, even in the later days of his illness; what would have been to most a depressing experience was transmuted by his spirit into yet another new adventure, that was to be savoured to the full, for it could only happen once.

In her long and intimate appreciation Lady Ridley touched on many facets of his work and character:

The antennae of his intuition were so exquisitely sensitive that it was easy for him to know what other people were feeling, and it was this quality that made him for many a great doctor and a great teacher, but equally his intuition would reveal to him those whom he could not help, and this resulted in his sometimes being misunderstood by his contemporaries as well as by his students . . . he remembered what he read, for one of his many inherent gifts was a prodigious memory. He continued all his life to read voraciously and widely but never systematically. He would jump from book to book, from fiction to poetry, essay to biography, following either a trend of thought from one author to another, or in pursuit of a personality or a subject suggested by the last book he had read.

He has been described as 'an artist who survived a scientific training', and he certainly had the perspicacity of the artist, revealing to us 'things we had passed a thousand times nor cared to see'. And he had the wisdom, humour and sweetness of a man who had come to terms with himself. But he could equally be described as a man of deep emotions which he continuously subjected to a scrutinising analysis.

And in the homage of his younger colleagues—those whom he had inevitably gathered about him during the growing years of the Department—there is a wellspring of devoted appreciation, understanding, and gratitude.

Dr. Fred Miller speaks of the personal meaning of the Department:

Teaching that the fundamental purpose of a university department is to advance knowledge of its subject, James Spence recognised that a child health department in the faculty of medicine has other responsibilities—to care for sick children; to teach undergraduates; to transmit changing knowledge to family doctors; and to undertake research with or on behalf of local health authorities. These were the objects of his department. The instrument to achieve these ends was a group of friends, with a similar basic philosophy of medicine and able to work and think together, who, sharing all the activities of teaching, clinical responsibility, and research would gradually build up a common body of knowledge and experience for the service of others. Such a group is not quickly formed; but as it increased, James Spence was always at the centre, accessible, stimulating, guiding, helping, sharing his wisdom generously. And always, despite his distant travels abroad, his frequent journeys south, and his opportunities for other and apparently greater responsibilities, he returned gladly and thankfully to Newcastle, confessing as all true Novocastrians do, that deep sense of homecoming as the train crosses the High Level Bridge, above the dark river with its twinkling lights, into the Central Station.

His conception of a department of friends working together for a common end was achieved for these few brief and happy years: it brought order and a sense of form and corporate spirit to the work—an order not perhaps apparent to all those inside the department, for it was never irksome, but seemingly obvious to many of our frequent visitors, who amused us by admiring what they called 'the organisation'. In the last few years we were perhaps from time to time jealous of the Medical Research Council, the University Grants Committee, and other bodies which claimed much of his time; but we realised they had more than a local significance, and we carried on as best we could, valuing more and more the moments we did spend with him. He has left a department of devoted people bound to him and to each other by years of comradeship. We know we have been privileged, we hope we shall be durable, retaining the lessons, the discipline and the tradition we have learnt, carrying on as he would have wished until we have occasion to remember that last lesson he taught. That death can be met without flinching even when it advances slowly.

And Dr. Donald Court, who succeeded James Spence in the Chair which he had inaugurated, writes in equally moving words:

But it was in the conception and creation of the university department that his sensitivity and strength found their fullest expression. The simple but profound conviction which guided his thinking in this field I first learned from the postscript to a card telling me the arrangements

for my coming to Newcastle. It ran as follows: 'The first aim of my department is comradeship not achievement.' Friendship between all concerned with the care of children in Newcastle was the catalyst which brought into being the combined medical and surgical units, the clear definition of the aims and instruments of undergraduate teaching, and the varied pattern of clinical and social investigation which he developed in the last years of his life. He was a great teacher of the gifted student; those who were seeking a main road to the final examination often found his exacting standard of clinical accuracy and his unpredictable questions too much for them. They recognised that 'despite the manner of a kindly patrician he was a dangerous man'. He was at his best when faced with the swift and calamitous illness in the ward, or painting a vivid picture of disease in the twenty minutes of a clinical lecture, and above all in sensing and handling the hidden fears of a mother in consultation. Indeed for him consultation was the fundamental technique of medical practice and he believed that a sound medical education would train a man, whether he became specialist or family doctor, in the 'art of consultation'.

He had too a love of the novel and the unexpected—new faces, new stories and new ideas—and a capacity for distilling their essence and adding it quickly to his current counsel which, though refreshing, sometimes led to a superficiality of judgement. Yet even when you felt sure that he was wrong, you always emerged wiser from the encounter. James Spence, artist and scientist, romantic and realist, conservative and rebel, was always a leader. 'He had novel things to say and said them with the civil assurance of the humane scholar speaking authoritatively on matters of weight'.

Leadership, that intangible and yet shining virtue, is one of the most potent gifts with which mortals can be endowed. It was given to James Spence, and rested lightly on his shoulders. As it is the lot of some to lead, and the many to follow with unspoken acceptance, so he led those who walked with him, easily, gently, and yet directly. Even now his influence, pervasive as always, is immanent and brings men to talk of him as if he were still with them, in the little room of his Department in Newcastle, or at the Athenaeum, or round the long table in the chamber overlooking St. James's Park, where the Medical Research Council meets month by month.

For those who know the story of the final months of James Spence's life, and the courage which he displayed in complete keeping with his character and history, there is a striking comparison to be drawn in the last chapter of Marius the Epicurean. And perhaps there is to be found his best epitaph. He was amongst

PART I

The Nature of Disease

plantar response on one side only, and it would appear that there was also some paralysis of the muscles of accommodation in the eyes. The second case had a paralytic squint. He had also nasal speech and palatal paralysis resembling diphtheritic palsy, yet in the absence of any evidence that it was diphtheritic in origin: a point to be noted, for we shall refer to a case of Bright's described in 1836, which appears to have shown the same disability. Another point to be noted, although its significance cannot be explained, is that the first case had a typical duodenal ulcer, a rare event in a boy of 9.

CASE 1. Boy aged 9 years. Admitted 19 April 1926. Died 22 April 1926

A very accurate and detailed history was obtained from Dr. Charles, who referred the case to hospital as follows: A perfectly healthy active boy up to onset of illness 3 weeks before admission. Member of a healthy family with no evidence of syphilis. The first symptoms were lassitude and drowsiness, with some vomiting in the first week. During the second week he was easily tired and peculiarly lethargic and drowsy, and the possibility of encephalitis or meningitis was considered. There were nausea and occasional vomiting with some slight abdominal pain and an attack of diarrhoea for a few days. At this stage his eyesight appears to have become affected in a peculiar way. He was not yet acutely ill, being able to sit about at the fireside, and he is described as being 'unable to see things properly and groping for his food at table'. In the third week he became worse and took to his bed. Two days before admission (17 April 1926) he was obviously dangerously ill with swollen abdomen, oedema of the feet, and delirium. At this time there was no jaundice, and the urine contained no albumen or sugar.

On admission on 19 April 1926 the boy was extremely ill and restless. Pulse 112. Respirations 24. Temperature normal. There was obvious recent loss of subcutaneous fat. There was considerable ascites, and the liver was smaller than normal though the edge of the left lobe could be felt high up in the epigastrium and was very hard. Spleen enlarged. The heart and lungs showed nothing to account for the illness. The urine was acid, 1.025, no albumen and contained a trace of sugar. Cerebro-spinal fluid was normal. Blood urea 40 mgm. per 100 c.c. No jaundice or petechiae present. Occult blood was present in the stools.

On this day the nervous signs were as follows: He was in a restless state suggesting at first sight tuberculous meningitis, but differing from that in the way he would sit up apparently quite conscious, giving vent to peculiar wails and piercing shrieks, refusing to be comforted. These were not mere complaints of acute pain or the insensate cries of semi-coma. They went on at times for as long as 10 or 15 minutes while he sat up in the corner of his cot, resembling some terror-stricken animal in its cage. He would then subside into quieter states of irritability or restlessness, to be followed again by these attacks of active shrieking and plaintive wailing. The pupils were widely dilated, but reacted to light. As far as could be seen there was no reaction to accommodation and the fundi oculi were normal. No squints or ptosis. Both knee-jerks and ankle-jerks were present. On the right side there was a well-marked extensor plantar response, on the left it was equivocal. There was no stiffness of the neck. During the next 3 days before death the extensor response on the right side persisted, the left plantar response remained indefinite. Pupils remained widely dilated. The restless state continued, but now the periods of crying and wailing were varied by the intermission of convulsive attacks in which he held himself in a position of opisthotonos, the attacks lasting perhaps half a minute and severe enough to suggest poisoning by strychnine. When these passed there was no rigidity and no neck stiffness, and he lay for the most part quiet and flaccid with occasional return of the 'cholaemic crying'. At this stage the Ward Sister ventured the opinion that in spite of the obvious resemblance to meningitis, she had never seen a case of meningitis behave as this case, in the way he changed from one state to another, nor had she ever heard such peculiar wailing going on as it did at intervals for 2 days and nights. On 22 April 1926 petechiae appeared on the arms and trunk. He vomited blood, then sank into a weak comatose state and died. Jaundice, though eagerly sought for, was absent throughout the illness until 24 hours before death when it appeared in the conjunctiva. Blood taken for a Van der Bergh test on the last day gave a direct positive reaction. A trace of sugar was present in the urine until the last day.

Post-mortem examination (by Dr. A. F. B. Shaw):

A slight degree of jaundice could be noticed in the conjunctiva and skin. A typical condition of advanced subacute atrophy of the

During these attacks he could not be roused and there was loss of sphincter control. Admitted 9 days before death there were no nervous symptoms or signs. Two days later he had an attack (the abdomen had been tapped a few hours before) in which he is described as 'very noisy for an hour or two, shouting and crying, and with incontinence of urine and faeces'. He recovered temporarily from this and later became unconscious and died.

Although the appearance of coma or noisy delirium usually denotes the approach of the terminal stage of the disease, we feel that it is neither necessary nor profitable to attempt to define them too closely as a ready means of foretelling the issue. Stuart M'Donald and Milne, in a study of the pathological changes in subacute atrophy of the liver, have commented on the difficulty of estimating the duration of the morbid processes. They describe cases in which they judged the liver changes to have been present for varying periods up to 7 months. The same difficulty exists in attempting to assess the significance of some of the vague symptoms such as lassitude and sleepiness which some of the cases show for several months before more definite evidence of liver disease shows itself. Moreover, this difficulty is presented more clearly when we remember that all studies of subacute or acute liver atrophy are based on cases which ultimately die. It seems to be tacitly understood that such a diagnosis is ventured only when the liver disturbances, or jaundice if it be present, are severe enough to kill the patient. Short of that, or if recovery takes place, the diagnosis of catarrhal jaundice or perhaps 'acidosis' is given. Apropos of this we have recently heard of a case, of which we are not able to give full details, in which the patient had symptoms suggesting encephalitis from which he recovered. For the next 6 months he had vague abdominal and digestive trouble for which a laparotomy was done. At the operation a 'cirrhotic condition of the liver' was found. This may be a case in which acute or subacute atrophy of the liver was present at the time of the encephalitic symptoms of lethargy and from which a recovery was made to go on later to cirrhosis. Byrom Bramwell's suggestion that certain unusual cases of cirrhosis in young people might be a phase of Wilson's disease in which the nervous manifestations had not yet declared themselves has a bearing on this point.

SUMMARY OF OBSERVATIONS

Without giving details of the other 3 cases in children and of the remaining adult cases, some points of interest in the whole series of 21 cases can be summarized for brief discussion.

Of 21 cases of liver atrophy 5 were children of 14 or under. These were traced from a total number of 4,958 post-mortems in a period of 13 years, and thus represent only the fatal cases which occurred in this time. The possibility exists that other cases have occurred and have recovered.

The pathological changes in the liver in these cases were as follows: 1 case of acute atrophy (acute yellow atrophy) and 20 cases of subacute yellow atrophy. Or classifying them in the way that Miller and Rutherford suggest, in which the subacute group is further subdivided according to the amount of regenerative changes found in the liver: 1 case of acute atrophy with early fibrosis, 5 cases of subacute atrophy with early fibrosis, 15 cases of subacute atrophy with marked nodular hyperplasia.

In all 5 cases of children, and in 11 of the adult cases, a certain sequence of nervous symptoms characterized the terminal or 'cholaemic' stage. This was more evident in the children than in the adults and consisted of lethargy or stupor, a period of unusual shrieking and crying, a stage of convulsions, death in coma.

The most characteristic of these stages of nervous symptoms was that of maniacal shrieking and wailing which we have termed 'cholaemic crying'. It was not present in all cases, but when it occurred it was distinctive, differing from mere toxic delirium.

In 8 cases where a systematic examination of the reflexes was recorded extensor plantar responses were present in 3. In 1 of these it was unilateral.

In 1 case there was palatal paralysis and nasal speech.

In 1 case there was a paralytic strabismus due to a sixth nerve paralysis.

In 4 cases cerebrospinal fluids were examined. In none of these were there increases of cells or of protein.

Jaundice was present in all cases but varied greatly in degree, and the appearance of the nervous cholaemic symptoms can bear no relation to it, when we consider the features of the first case we have described. For there a slight tinge of jaundice appeared only in the last 24 hours, when the boy had already had acute nervous

symptoms of cholaemia for 6 days. (We recognize from this the mistaken etymology of the term 'cholaemia' in the sense that we have used it. But the use of it as a clinical term to express the terminal stage is justified, just as 'uraemia' is used for the analogous toxic stage in kidney disease, although excess of urea in the blood is not the toxin concerned.)

In 4 of the cases of the subacute type with ascites and jaundice, the acute cholaemic stage was precipitated by tapping the abdomen to relieve abdominal discomfort.

A terminal pyrexia was noted in 5 cases, only 2 of which had broncho-pneumonia. Thus the nervous symptoms of cholaemia cannot be explained by a sudden accession of fever.

In 6 cases there was evidence of syphilis and treatment by salvarsan, and in 1 other case of congenital syphilis without treatment by salvarsan. The clinical picture of the cholaemic stage in this group did not differ from that of the other cases.

DISCUSSION

A few of these points will need more discussion than the comment we have made on them.

Extensor plantar responses

It has long been established that these may occur in cholaemia. As far as we can find Rolleston first drew attention to it in describing a case of acute yellow atrophy. Later Willcox in his Lettsonian lectures described cases of infective jaundice, and Elliott and Walshe cases of hepatic cirrhosis, which developed extensor plantar responses in the terminal cholaemic stage. These are mentioned again by Walshe in his review 'The Babinski plantar response in toxic states'. Elliott and Walshe state that the stage of cholaemia may be recognized 'by the mental changes of mild delirium or drowsiness, associated with a double extensor response'. This suggests that the extensor plantar response characterizes the toxic stage of cholaemia and is a necessary sign or accompaniment of it, for they go on to state the converse that other cases of hepatic disease may sink 'quietly and slowly to death without acute mental changes and without an extensor response even in the last hours, that is, without cholaemia'. We agree that careful and repeated examinations of the plantar reflexes should be made in any

suspected case, but we cannot agree that when cholaemia ensues, extensor responses will necessarily be found. In our first case it was present on the right side and indefinite on the left. But in the second the responses remained definitely flexor, for they were tested carefully and repeated up to the hour of death. Yet this case had the other classical symptoms of cholaemia; maniacal shrieking, jactitations, and fits passing on to coma and death. We must therefore presume that although extensor responses may be a valuable physical sign of cholaemia, they are not necessarily present.

Palatal paralysis

This occurred with well-defined nasal speech in 1 case. It was observed in the early stage for a day or two preceding the maniacal shouting, convulsions, or coma, and the lack of movement of the palate was easily demonstrable. Its appearance may have been quite fortuitous in our case. We therefore hesitate to define it as a nervous sign of cholaemia, but on the other hand it may be so. In the case described there was no evidence of any other possible cause such as diphtheria.

We have been unable to find any record of it in any previously reported case, unless it be a short note that Richard Bright made on one of his cases. This was in 1836 in a paper which he wrote in the first volume of the *Guy's Hospital Reports* on 'Observations on Jaundice: more particularly on that form which accompanies the diffused inflammation of the substance of the liver'. In this he describes graphically the clinical picture of the condition we now term acute liver atrophy. The note was made 6 days before death and reads: 'She generally prefers the sitting posture in bed. Some sluggishness of speech and a plaintive tone.'

Maniacal shouting and convulsions

The maniacal shouting (cholaemic crying) was more evident in the children than in the adults. They gave the appearance of suffering intense pain which they could not explain and it is of interest that we noted that when the shouting and crying went on they 'preferred the sitting posture', as Bright describes it. As a reference to other descriptions of the noisiness, restlessness, and convulsions we can do no better than quote further from Bright. Of one of his cases he writes: 'She had all the appearance of a person sinking from the loss of blood, with constant and distressing

jactitations, and delirious though faint exclamations. She continued delirious and sank in the afternoon of the following day.' Of another: 'Nov. 28th.—She generally prefers the sitting posture in bed. Some sluggishness in her mode of speech, and a plaintive tone. Dec. 1st.—Her pupils are rather dilated; her mode of utterance is dull and indistinct; complains of loss of power in the left hand; the right is already disabled by disease. Dec. 2nd.—Is lying on her right side, drowsy, with her legs drawn up, moving her left hand with a kind of jactitation. Dec. 3rd.—Yesterday evening she was screaming loudly, with her tongue protruded beyond her teeth. Today she is in a state of perfect coma with the eyes turned up.' And of another: 'She lay in a perfectly torpid state the whole night; but towards the morning became delirious, so that it was with difficulty she could be restrained in her bed. At the time of the visit she was very restless, and seemed to suffer pain; but, was unable to answer questions.'

Cause of cholaemia

Of the ultimate cause of the toxic state in liver diseases which we term cholaemia, we can offer no explanation. It would appear that the phrase 'hepatic insufficiency' is inadequate to do this. This implies that a stage of destruction of liver tissue is reached when the hepatic functions fail altogether and toxæmia ensues. Following the results of the experimental Eck's fistula in animals in which convulsions ensued after administration of protein but not of carbohydrate or fat, it has been held that the symptoms of cholaemia are due to the passage of protein substances through the damaged liver that have not been de-aminised. In 3 of our cases only were estimations made of the blood urea or urine urea. These were carried out at the height of cholaemia and yet the percentage of urine urea reached 2.4 per cent. to 3.0 per cent. and the blood urea was normal. This hardly suggests a complete breakdown of the urea-forming function of the liver. Moreover, cholaemia may arise as readily in the subacute cases where rapid regeneration of liver tissue has taken place as in the acute cases with extensive destructive changes. The researches of Mann and others at first suggest hypoglycaemia as the cause of the cholæmic convulsions. They found a progressive fall of blood-sugar, ending in convulsions and death, in dogs whose livers they had successfully extirpated. This explanation, however, will not serve in our first 2 cases. In

both, traces of sugar were found in the urine, and in one the blood-sugar was high (0.226 per cent.). Another possible explanation that could be offered is the production of some autolytic poison from the liver substance itself at some stage or other of the pathological changes which it undergoes. This is suggested by a case which one of us saw 6 years ago. It was a boy with lymphadenoma whose enlarged cervical glands were being treated by X-rays. The glands began to decrease in size, but in the course of treatment he developed severe toxic symptoms which lasted 3 to 4 days, and resembled closely the picture we have drawn of cholaemia. This case was fully investigated at the time and a careful post-mortem examination made, but none of the usual causes of terminal toxæmia were found, and we were left to presume that the condition had resulted from the release of some autolytic toxin from the lymphadenomatous glands.

Our thanks are due to Professor Stuart M'Donald for the use of the pathological records and for help in re-examination of the microscopic slides of all these cases, and to the members of the Honorary Staff of the Hospital whose clinical records we have used for this paper.

REFERENCES

- BRADWELL, B. 1916. *Edinb. med. J.* 17, (N.S.), 1916, p. 90.
 BRIGHT, R. 1836. *Guy's Hosp. Rep.* 1, 1936, p. 604.
 ELLIOTT, T. R., and WALSH, F. M. R. 1925. *Lancet*, 208, 1925, p. 65.
 M'DONALD, S., and MILNE, L. S. 1909. *J. Path. Bact.*, 13, 1909, p. 161.
 MANN, F. C. 1921. *Amer. J. Physiol.* 55, 1921, p. 285.
 ——— 1922. *Ibid.* 59, 1922, p. 484.
 ——— 1922-3. *Ibid.* 63, 1922-3, pp. 397, 424.
 MILLER, J., and RUTHERFORD, A. 1923. *Quart. J. Med.* 17, 1923, p. 81.
 ROLLESTON, H. D. 1912. *Diseases of the Liver, Gall-bladder and Bile-ducts*, 1912, p. 582.
 WALSH, F. M. R. 1925. *Med. Sci. Abstr.* 12, 1925, p. 261.
 WILLCOX, W. H. 1919. *Trans. med. Soc. Lond.* 42, 1919, p. 65.

II. *The Liver and Pernicious Anaemia*¹

MINOT and Murphy of Boston and their colleagues have shown that there is something in mammalian liver which, taken by the mouth, is effective in the treatment of pernicious anaemia. The work by means of which they reached this discovery is a beautiful example of clinical study and research, and deserves close attention. As far as can be gathered its origin lay in their hypothesis that pernicious anaemia might be a deficiency disease and due to some fault in diet. This led them to treat a number of cases on various diets, and in 1926 they were able to publish successful results of the treatment of 45 cases by a special diet. This diet was made up of liberal amounts of liver, meat, vegetables, and fruit, with a restriction of fat and carbohydrate. Later, it was revealed that the essential curative factor was the liver, and that the other elements of the diet had but little influence. The research was pursued still further with the help of Cohn, on the lines of dividing the liver into various fractions by a process of discarding those portions which were found to be inactive, and subdividing the active portions. Until now the stage has been reached where they have shown that the essential curative factor is probably a 'liver extract'. This is a certain non-protein fraction which represents about 1 per cent. of the liver, a few grammes of which act with apparent specificity in pernicious anaemia when taken by the mouth each day. It is already obvious that this discovery has a greater significance than the mere provision of a cure for pernicious anaemia; for it will probably throw fresh light on physiological processes, both in the function of the liver and in the generation of red blood cells, which have not been recognized hitherto.

The clinical results of Minot and Murphy's cases are now widely known and have received ample confirmation. It will suffice to mention briefly their latest figures, published in the *British Medical Journal* of 15 October 1927 (p. 674). There they report the results of 125 cases which have been treated with regularity for from

¹ Read before the Newcastle and Northern Counties Medical Society on 3 November 1927. Published in *Newcastle Medical Journal*, 5, 1927, p. 71.

TABLE 2

*Erythrocyte counts in 20 cases of pernicious anaemia
treated with liver diet or liver extract*

Red cells in millions

	<i>Before</i>	<i>1 wks.</i>	<i>2 wks.</i>	<i>3 wks.</i>	<i>4 wks.</i>	<i>2 mths.</i>	<i>3 mths.</i>	<i>4 mths.</i>	<i>6 mths.</i>
1 Fr.	1.2 (3)	5.12
2 Di.	1.2 (4)	1.3	1.4	3.1	4.0	5.1
3 Li.	2.1	..	4.4	..	4.5
4 Gi.	0.70 (2)	1.5	2.0	..	5.0
5 Pa.	2.5	..	3.0	4.3
6 Gr.	1.0	3.8	4.0	5.0
7 M.E.	1.6	3.4	..	3.5	4.0	5.0
8 Do.	1.7	..	3.0	..	4.5
9 Te.	3.4	..	5.5
10 Ma.	1.3	..	5.3	..	5.5	6.0
11 Te.	1.0	..	1.8	3.5	4.4	4.5
12 Ba.	1.2	1.2	1.2	1.9	5.5	6.5	4.2
13 Lo.	2.0	..	3.4	..	3.5	4.0
14 Bu.	0.9	..	1.4	..	1.6	3.5	4.5
15 Pu.	3.1	4.0	4.5
16 Ho.	3.2	2.9	2.6	1.7
17 Cr.	0.8	0.9	1.7	2.2	3.5	4.5
18 Do.	1.0	4.5
19 Bo.	1.2	..	2.0	2.30	..	4.0
20 Ni.	1.4	2.7	4.4	4.9	6.30

count was 1,600,000. After admission to hospital he was placed out of doors and treated with arsenic. After 10 weeks his red count was 5,350,000. But it is common experience that this will probably be only a temporary improvement, and that a relapse will occur within a few months. It is of interest that this man, in spite of the rise in red cells, did not experience the feeling of improved general health and well-being so rapidly or in so marked a degree as the liver-treated patients.

I have made a statistical comparison of cases treated with liver and cases treated without liver. This is shown in Fig. 1. Only the severest cases of pernicious anaemia with red counts below 2 million have been taken. Eleven cases treated with liver, having an average initial count of 1,100,000 red cells, reached a level of nearly 5 million in 2 months. Ten cases having other methods of treatment, whose results I have taken from the hospital's records, showed only a rise from $1\frac{1}{2}$ millions to $2\frac{1}{2}$ millions in the same period. We have not been able to follow our cases over any great

length of time, but I think that so far we have evidence which indicates that the liver treatment of pernicious anaemia causes a more rapid and successful rise of red blood cells than any other form of treatment. This confirms the discovery of Minot and Murphy.

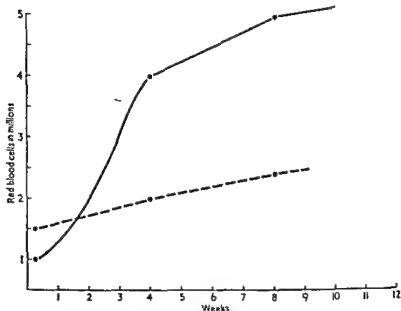


FIG. 1. The average rise of red blood cells in 11 treated cases with initial count below 2 millions (10 control cases without liver).

General symptoms

I need say little about the improvement in the general condition of the successfully treated cases. Some of the severest of these have been able to resume full activities at the end of 3 months, professing to feel quite fit. But with regard to individual symptoms of the disease I have been struck with the rapidity with which the tongue symptoms may disappear. In one case of a woman, aged 55, with typical glossitis and a red count below $1\frac{1}{2}$ millions, there had been most pronounced soreness of the tongue for several months which had prevented her from taking any salty or bitter food. Within 6 days of commencing treatment, which in this instance was the giving of 20 grammes of a liver extract daily, all soreness of the

tongue had disappeared before there had been any rise in the red cells or haemoglobin, or any improvement in her general condition. Her later progress was uneventfully successful.

Two of the cases while under treatment have had acute attacks of gout. Another has developed marked oedema of the legs, which is apparently due to venous thrombosis. It is significant that these cases had eaten liver very freely and probably more than a pound a day. Also in 2 of them there had been a very rapid rise of red cells to a level of 6 millions. This suggests that the response to the diet is a quantitative one, dependent on the amount of liver taken. Attention will have to be paid to this point in the future. Already we have found that once the red cells have reached a level of $4\frac{1}{2}$ or 5 millions no more liver should be taken than is necessary to maintain that level. In practice this maintenance amount will probably be about $\frac{1}{2}$ lb. of liver four times a week.

The reticulocyte crisis

Most of the advances in medicine are made by the intelligent use of some new method of investigation, and Minot and Murphy's work on pernicious anaemia has been greatly helped by making use of the estimation of the number of reticulated red cells in the blood, which can be revealed by certain methods of vital staining. It has been shown that these cells are present in normal blood in the proportion of about 2 to 5 per cent. of the total red cells. In cases of untreated pernicious anaemia they may be lower still. Their presence can be taken as evidence of the formation of mature and normal red cells. If and when a sudden formation of mature red cells takes place, and these are thrown into the blood-stream, there is an immediate rise of the proportion of reticulocytes for a few days. This apparently is what happens when a case of pernicious anaemia is given a quickly curative treatment such as a liver diet. Where there is a very low blood cell count the response to treatment may be sufficiently effective to cause an appearance of reticulated red cells in the blood, in proportions of 40 or even 50 per cent., lasting a few days. This sudden rise is spoken of as the 'reticulocyte crisis'. It usually reaches its climax within 7 days. This may be considered to be only of academic interest, but it is important as a rapid method of estimating the efficacy of treatment. And it has been used as such in testing the various fractions of liver which Cohn and his colleagues have made. If, for example,

a certain fraction of liver were given to a case of pernicious anaemia with a very low blood count, and no reticulocyte crisis occurred, it could be concluded that this fraction was ineffective. It is in this way that the later steps of Minot and Murphy's researches have been helped and hastened. But it must be remembered that a brisk reticulocyte crisis is to be expected only in those cases with

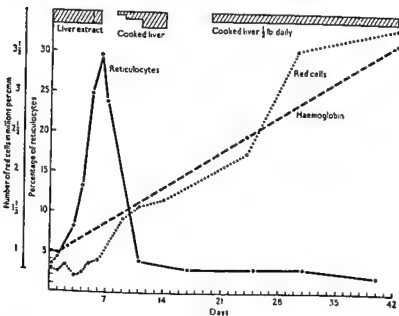


FIG. 2. Reticulocyte crisis and the rise of red cells during treatment (case No. 17).

low initial red counts who, without previously having had any treatment, are placed on a sufficient dose of liver to provoke an immediate maturation and release of new red cells from the bone marrow. When once this process is set up the total number of red cells begins to rise and the reticulocytes return to a steady level of 3 to 5 per cent. Fig. 2 illustrates an example of this reticulocyte crisis and steady rise of red cells during treatment.

The case (No. 17) was a woman, aged 60, who had been under observation and treatment for pernicious anaemia for 2 years. The diagnosis had been carefully established during her earlier

apathetic. She can walk with ease and her appetite has returned so that she really enjoys her food.'

The liver extract

In an attempt to find some concentrated form of liver which could be given more easily than liver itself, we first made use of some 'compressed liver tablets' which are placed on the market by a proprietary firm. Two cases were tried on these, but it was found that they had no effect whatsoever in the doses advocated on the label of the bottle. The dose was increased until instead of 3 tablets, 100 tablets a day were taken. A slight response was obtained in 1 patient. But it was not equivalent to the response given by $\frac{1}{2}$ lb. of lightly cooked liver. Moreover the cost of this therapy was more than twenty shillings a day for each patient, so it was discontinued.

After the publication of the paper of Cohn and his colleagues on 'The nature of the material in liver effective in pernicious anaemia' we attempted to make a concentrated liver fraction in the manner which they described. I need not here refer to the technical details and difficulties of the process. But it must be admitted that the first batch that was made appeared to have little effect. We repeated it again, taking more liver and substituting another method of precipitating the protein. This was found to be more successful. To one patient (No. 17 which I have already described) 30 grammes were given daily for 6 days, and the response was definite with a reticulocyte crisis of 30 per cent. To another patient from 20 to 25 grammes were given daily for 3 weeks. Here again a similar response was obtained. After a reticulocyte crisis of 28 per cent. the red cells rose from an initial count of 1,200,000 to 2,300,000 in 3 weeks with very definite improvement of her general condition. Here again we were at the end of our resources, having no more extract ready for use.

In the process of the preparation of this extract we found that we were losing a considerable portion of the effective material of the liver. And as far as can be told from our few experiments it appears that it required extract which was obtained from $2\frac{1}{2}$ lb. of liver before an effect equivalent to that of 1 lb. of lightly cooked liver was obtained. That is that in its preparation more than half of the effective material was lost. I cannot yet say whether this was destroyed in the preparation, or lost through incomplete washings of the acid precipitable and heat coagulable protein precipitates

which are discarded. But even so, 25 grammes of extract from 2½ lb. of liver is much more easily eaten than a pound of liver itself, so a very practical advance may be expected from this work which Cohn and others are doing.

The discovery of an extract of liver which is effective in the cure of pernicious anaemia naturally leads to some speculation on the etiology of the disease. Probably it will lead to a revision of the recently prevalent ideas on the subject. At first sight it suggests that the disease is a disorder of an internal secretion of the liver. But further work must be done to prove it. But I would suggest that Peabody's paper 'On the pathology of the bone marrow in pernicious anaemia' be read. He describes observations he has made on the bone marrow of patients obtained by punctures before and during treatment with liver. His work appears to me to dispose of the theory that pernicious anaemia is a result of a primarily haemolytic process. Probably the haemolysis which occurs in this disease is purely a secondary factor which ensues when immature and functionally inefficient red cells are thrust into the bloodstream.

The observations that have been made for this paper could not have been gathered without the enthusiastic assistance given by the House Physicians, Drs. D. S. Jackson, M. M. Suzman, G. Y. Feggetter, and C. C. Ungley, to whom most credit is due.

REFERENCES

- COHN, E. J., and others. 1927. *J. biol. chem.* 74, 1927, p. lxix.
MINOT, G. R., and MURPHY, W. P. 1926. *J. Amer. med. Ass.* 87, 1926, p. 470.
——— 1927. *Ibid.* 89, 1927, p. 759.
PEABODY, F. W. 1927. *Amer. J. Path.* 3, 1927, p. 179.

III. *A Clinical Study of Nutritional Xerophthalmia and Night-Blindness*¹

OVER a period of 12 months 17 cases of nutritional xerophthalmia or night-blindness have been collected and studied. In doing this I have made observations on the incidence and treatment of the disease, and also investigated the association of xerophthalmia with a lowered resistance to general infections, with the rate of growth, and with evidence of damage to epithelial structures in other parts of the body.

It is now accepted that a specific diet deficiency, probably an absence of Vitamin A, will produce the disease, and that the symptoms of it in order of severity will be: (1) night-blindness, (2) xerosis conjunctivae with the characteristic whitish deposits on the scleral conjunctivae known as Bitot's spots; and (3) keratomalacia. In patients slightly affected, especially in adults, there may appear night-blindness only without xerophthalmia. And the younger the patient the more probability there is of severe keratomalacia.

The affected patients were all past the age of infancy, the youngest being 2 years old. There was no special age incidence, but 11 of them were children of 14 years or under. There was a history of an evidently deficient diet, with little or no animal fat or meat protein. Two cases, however, had eaten an egg every day without preventing the onset of the disease. There was no definite seasonal incidence in the *onset* of the disease. Night-blindness became accentuated in the spring and summer months, but the investigation suggested that this was due to the extra physical exertion from games or work at this time of the year, and not to exposure to bright sunshine.

In all the older patients night-blindness was the first and chief complaint. In 3 of these there were no other signs. In all others there was xerosis conjunctivae, Bitot's spots, or commencing keratomalacia. In the two youngest patients, both aged 2 years, there was no complaint of night-blindness, but it was probably present.

¹ From the Transactions of the Second International Paediatric Congress. Published in *Acta Paediatrica*, 11, 1930, p. 541.

All cases under the age of 12 were carefully examined clinically and radiographically for signs of rickets both before and after treatment. In 3 cases, aged 2, 4, and 12, blood calcium and phosphorus estimations were also done. The general condition of all the patients was relatively good. There was in no case evidence of rickets, scurvy, beri-beri, pellagra, or oedema.

There had been no obvious cessation of growth in the xerophthalmic children while on the deficient diet. With the cure of the xerophthalmia there was no striking rise of the weight and height curve as happens in experimental animals.

An investigation of the families of the 11 youngest cases showed that there had been no unusual incidence of general infections or lowered resistance to them. Amongst 99 people in these families during a period of 2 years there had been 1 death from pneumonia, 2 cases of mumps, and 3 cases of measles with complete recovery. There was, however, a very high incidence of skin sepsis, impetigo, and boils. This was cured quickly by anti-xerophthalmic treatment.

The amount of epithelial cells in the urine of the xerophthalmic patients was one of the most striking features of the disease. It is probably a characteristic phenomenon of this form of diet deficiency.

In the treatment of the cases a study was made of the maximum rate of cure with cod-liver oil, butter, and milk. With this as a basis of comparison it was shown that in 1 case dried liver extract, effective in pernicious anaemia, had no curative effect in xerophthalmia. In 2 cases irradiated Ergosterol had no curative effect. In another case exposure to ultra-violet radiation had no curative effect.

In summary my investigation suggests (1) the great significance of skin infections and epithelial cells in the urine as evidence of xerophthalmic diet deficiency, and (2) the specific food factor (probably Vitamin A) deficient in this diet is not the same as the growth factor or as the anti-infection factor for general infections.

IV. Benign Tuberculous Infiltration of the Lung (*Epituberculosis*)¹

THE following observations may throw some light upon the condition described as epituberculous infiltration or epituberculosis, a condition which probably is not different from that formerly described as tuberculous spleno-pneumonia, *Frühinfiltrat*, or *tuberculose inflammatoire*.

It is difficult to define exactly the term 'epituberculous infiltration' for it is used to denote a variable clinical picture, of which the underlying pathology is not known through lack of opportunity for post-mortem study. It was first used in 1920 by Eliasberg and Neuland to describe a benign clinical syndrome in a tuberculous child, of which the chief features were an extensive dense consolidation of the lung revealed by percussion, auscultation, and X-ray examination; a harsh cough without sputum; an absence of severe constitutional symptoms; an absence of an initial acute illness; a slow disappearance of the consolidation, without signs of softening or cavitation; a complete recovery without fibrosis or bronchiectasis. The nature of the lesion is considered by many to be an allergic reaction around a small primary tuberculous focus, but this has been deduced by speculation and not proved by pathological investigation. The argument that has been advanced in favour of this theory is that, were the whole lesion a caseous tuberculous process or gelatinous pneumonia, the illness would be more severe, the course of it less benign, and the result less satisfactory.

The 3 cases to be described had that combination of an extensive solid lesion of the lung, with relatively slight symptoms of illness, which justifies their being regarded as examples of children suffering from epituberculous infiltration of the lung. They were presumably tuberculous children, the skin tuberculin reaction being positive, and in 2 there was a definite history of contact. From none of them were tubercle bacilli recovered in the sputum or stomach washings. In the third a further investigation was pursued by exploring the lung lesion and examining the material obtained

¹ Published in *Archives of Diseases in Childhood*, 7, no. 37, February 1932, p. 1.

in the aperture of the exploring needle. This was done twice at an interval of 4 weeks, and in two sites. On both occasions tubercle bacilli were present in the material. This placed the tuberculous nature of the process in this case beyond doubt and the evidence suggests that the whole lesion in so-called epituberculous infiltration is a caseous tuberculous process, yet producing no severe constitutional symptoms.

CASE REPORTS

The details of the 3 cases on which this communication is based are as follows:

CASE 1. A girl, R. G., 6 months old, came for the first time under observation on 14 November 1927. She had not gained weight for 2 months and it was for this reason that advice was sought. For nearly 3 months the child had been coughing but there had been no other symptoms.

The father was a man of 21 who had been in poor health for a few years. On examination it was found that he had active pulmonary tuberculosis with tubercle bacilli in the sputum. The mother was healthy. There had been 1 other child, who had died a year earlier of tuberculous meningitis.

The child was pallid, thin, and weighing 10 lb. She was not ill. She looked contented and happy, and the face itself was fat. On examination there was a pronounced dullness of the right thorax from the apex to the third intercostal space in front, and to the mid-scapular level behind. The note was equally dull in the upper part of the axilla. Over this affected area the respiratory murmur was diminished to a degree suggesting a pleural effusion. Distant tubular breathing could be heard, but there were no râles or rhonchi. At the base of the lung the percussion note and air entry were normal. The signs over the left lung were normal.

The abdomen was not protuberant: liver and spleen not enlarged. The urine was free from cells, albumin, and sugar. The heart was normal and not displaced. The skin, glands, mouth, and nasopharynx were normal. Haemoglobin = 65 per cent. Rectal temperature normal. Skin tuberculin reaction to 0.1 c.cm. of a 1 in 1,000 dilution was strongly positive, the reaction lasting 10 days.

The existence of a localized empyema was considered possible, so an exploring needle was put into the second interspace in front





(a) CASE 1. (2 February 1928) Radiograph taken 3 months after admission, showing the lung shadow as it persisted for the first 4 months



(b) CASE 1. (11 August 1928) Radiograph taken 6 months later



(c) CASE 1. (4 September 1929) Radiograph taken a year after discharge from hospital

and behind. No pus or fluid was found. A small amount of caseous material was present in the aperture of the needle's point, but this was not examined microscopically.

The child was kept in hospital for 10 months and remained happy and contented without symptoms of illness. For the first 3 weeks the temperature was normal. Then followed a bout of fever rising to 102° for 10 days. There was no alteration in the signs in the lung during this fever. Apart from this the child was afebrile throughout her stay, except for two or three rises of temperature for single days which were attributed to ward infections. There was no gain in weight for the first 3 months. Thereafter there was a steady gain. At the age of 12 months the weight was 12 lb.; at 15 months $14\frac{1}{2}$ lb.; at 16 months $15\frac{1}{2}$ lb.

The signs in the lung remained unaltered during her first 3 months in hospital. In February 1928 (child then 9 months) the dullness of the percussion note over the right upper lobe began to diminish, and the respiratory murmur became more pronounced. This improvement continued. A slightly impaired percussion note could still be detected in September 1928 (age 16 months) when the child was discharged, but the air entry to the upper lobe was normal.

The skin tuberculin reaction was repeated three times with 0.1 c.cm. of a 1 in 10,000 dilution and on each occasion gave a positive reaction of several days' duration.

On discharge the child was small and underweight, but was well covered with subcutaneous fat, and looked perfectly healthy. A year later, at the age of 2 years 4 months, she was in good health, weighed $21\frac{1}{2}$ lb., and was without cough. There was no splenomegaly, and no signs of disease could be elicited by auscultation or percussion.

In May 1930, at the age of 3, she was a healthy looking child of 26 lb. without sign or symptom of disease. In November 1931, at the age of $4\frac{1}{2}$ years, she was again in good health and appeared to be completely cured.

A series of X-ray examinations was made and those reproduced in Plate 1 are chosen from these to illustrate the X-ray appearance of the lesion, and the manner and rate of its disappearance. After admission the size and density of the shadow remained unaltered for about 4 months. Plate 1(a), taken on 2 February 1928, shows it at this stage. Plate 1(b) shows the shadow as it was on 11 August

1928, 9 months after admission; Plate 1(c) on 4 September 1929, a year after discharge. A later picture, taken in June 1931, shows that the shadow has completely disappeared. The consolidation lasted for about 2 years. When disappearing it receded first from the periphery of the lung.

CASE 2. E. T., a girl aged 5 years, admitted to hospital on 5 March 1930. She first attended as an out-patient with the complaint of lassitude and pallor. She had had no recent acute illnesses.

In the family there was no definite evidence of tuberculous infection, but a younger child aged $1\frac{1}{2}$ years had died some time before of meningitis believed to have been tuberculous.

The child was small but fairly well nourished. Weight $28\frac{1}{2}$ lb. She had no apparent symptoms and co-operated cheerfully and readily in the examination. She looked so healthy that it was surprising to find signs of a dull area over the whole of the upper part of the right lung. This was evident both in front and behind, and the signs of dullness with suppression of breath sounds suggested a localized pleural exudate. The area was explored by a needle and no fluid or pus obtained. The skin tuberculin reaction was strongly positive. X-ray examination revealed a dense area corresponding to almost the whole of the upper lobe with the lower limit sharply defined in its outer part (see Plate 2(a)).

From March 1930 until January 1932 she was kept under constant observation as an in-patient of the hospital, and later of Stannington sanatorium under the care of Dr. T. C. Hunter. During this time she continued in good health. There was no fever whatsoever: no cough and no sputum. She gained weight, and at the age of 7 she weighed 41 lb.

The consolidation of the right upper lobe began to disappear about 3 months after coming under observation. The rate of disappearance was slow, signs of dullness being still evident for more than a year. At no time was there any softening of the focus or evidence of cavitation. By January 1932 all signs had disappeared and she looked in perfect health.

A series of X-ray examinations showed a slow diminution in the size of the shadow receding from the outer margin of the lung (see Plate 2). The last of these (2(d)), taken on 30 October 1931, shows only an increased hilum shadow on the right side compared with that on the left.



(a) Case 2. (17 March 1930) Radiograph
taken shortly after admission



(c) Case 2. (27 October 1930) Radiograph
3 months after (b)



(b) Case 2. (23 July 1930) Radiograph
4 months later





(a) CASE 3. (8 July 1931) Radiograph taken shortly after admission, aged 7 months



(b) CASE 3. (19 October 1931) Radiograph taken 3 months later: patient afebrile and gaining weight

The signs and X-ray appearance of consolidation in the lung were in this child very similar to those in the first case, but disappeared more slowly.

CASE 3. A boy, W. S., aged 7 months, admitted to hospital on 3 July 1931. Brought for advice because of dyspepsia and failure to gain weight.

The mother died of pulmonary tuberculosis 9 weeks before the child was admitted. She had been in daily contact with him for the first 3 months of his life, and during this time she had had fever, with abundant sputum, containing tubercle bacilli. The father was apparently healthy. There were 4 other living children aged 14, 13, 11, and 6 years. A child aged 2 died at about the time of the birth of this child. The cause of death was unknown.

The child was small and underweight, 9½ lb. He was lively and contented. The abdomen was protuberant, but not distended. His appearance suggested a condition of dyspepsia due to lack of care and proper feeding.

On examination pronounced dullness was found over the upper part of the left lung. This was very evident in front and in the axilla, and less pronounced behind. The area of dullness extended down to the nipple. The breath sounds were diminished in that area and there was complete absence of crepitations and râles. On listening to the forced respiration provoked after crying slight wheezing rhonchi could be heard distantly, otherwise there were no adventitious sounds.

The right lung was normal. The heart was not displaced. The spleen was enlarged two fingers' breadth before the costal margin. Liver not enlarged. The skin was moist and clear. Glands palpable on both sides of the neck down the sterno-mastoids. Mouth and throat clean.

The temperature was normal. The urine contained a faint trace of albumin but no cells.

Skin tuberculin reaction was strongly positive to 0.1 c.cm. of 1 in 1,000 dilution. Haemoglobin 60 per cent. Leucocytes = 11,600.

The X-ray examination (Dr. Whately Davidson) on 8 July 1931 showed a dense shadow of the upper and middle part of the left lung (Plate 3(a)). The shadow diminished in intensity at the apex and the base was clear. The diaphragm moved normally.

A diagnosis of tuberculous infiltration of the lung was made.

For the next 2 months there was a rapid improvement in his general condition but the signs in the chest were unaltered. A note made on 4 September 1931 reads: 'The signs and symptoms are those of a so-called epituberculosis. He has been in hospital 2 months and gained 3 lb. Still underweight, but quite happy and symptomless. He feeds well, smiles and is contented. Temperature usually below 99°, and occasionally reaches 100°, but no bouts of fever. There is no cough, but Sister has noted periods of slight wheeziness lasting an hour or two. In spite of the absence of symptoms a big massive lesion persists in the upper part of the left lung, unaltered since admission.'

On 7 September the chest was explored. A needle was inserted into the third interspace in the mid-axillary line. No pus or fluid was obtained. A small amount of caseous material obtained in the needle was collected for examination. Two slide preparations were made. Each contained tubercle bacilli. A guinea-pig was inoculated with the material. Killed 5 weeks later it showed typical tuberculous lesions from which the bacilli were again recovered.

Four weeks later, on 9 October 1931, a second exploration was performed. The needle was introduced in front in the mid-clavicular line in the third interspace and directed medially, so that the point of contact with the lung should be as far removed as possible from the site of the previous puncture. No pus or fluid was obtained. A small amount of necrotic material was again found in the needle. This was examined in two slide preparations and again tubercle bacilli were present.

The child has remained in hospital and has now been under observation for 6 months. The present weight (6 January 1932) is 13 lb. 2 oz. During the past 6 weeks the weight has been stationary. Splenomegaly persists. The general condition improves. In November and December (fifth and sixth months of sojourn in hospital) the glands in the neck increased in size, but these are now subsiding.

A second X-ray examination (Plate 3(b)), on 19 October 1931, shows the dense shadow persisting, but diminishing in size, receding from the periphery. A third X-ray, on 6 January 1932, shows no further change.

DISCUSSION

The chief point of interest in these 3 cases is that clinically they resembled each other in being tuberculous children, each with a

massive lesion of the lung which was benign in character and lasted for a year or more, without much disturbance of the general health beyond some pallor and loss of weight in the early stages.

In the first 2 the evidence that the lesion was tuberculous was only presumptive. In favour of this view there are the facts that both had positive skin tuberculin reactions, and 1 a history of contact with a tuberculous parent. Any other explanation of the nature of the consolidation is difficult. The children had not had pneumonia, and the complete disappearance of the lesion after it had been present for a year, and the recovery of the lung without signs of fibrosis or bronchiectasis, exclude bronchial obstruction as the cause.

Görter and Lignac have published a paper under the title 'Collateral inflammation of the lung round a tuberculous centre' in which they report the case of a girl of 3 years with a dense shadow in the right lung from which they recovered tubercle bacilli by lung puncture. They were able to obtain a post-mortem examination 18 months later, by which time the dense lesion had greatly decreased in size. They put forward the view that the most probable explanation of the process was that from a small caseous focus there spread a collateral inflammation over the largest part of the affected lobe, and that an acute infection with pneumococci played an important role in this process. But in the present cases there was nothing to suggest or prove a recent pneumococcal infection.

The third case recorded above was infected at a very early age, shortly after birth, so the failure to gain weight in the first 6 months of his life had had a more evident effect on his general condition. Otherwise the physical signs of the lesion and his progress resembled those in the first 2 cases. In this third case the tuberculous nature of the process has been proved beyond doubt by recovery of the tubercle bacilli directly from the lung at two sites and on two occasions. The first exploration was made at a point which might conceivably have been the primary tuberculous focus, small in size and central in site. The second exploration was made at a point near the periphery of the consolidated area, as far as possible from the point of the first exploration. Tubercle bacilli were obtained in equal numbers and with equal facility from both parts of the lesion. It is clear, therefore, that in this case we have an infant infected shortly after birth with a large tuberculous lesion affecting almost the whole of the upper half of the lung, yet causing com-

paratively little disturbance to his health and no fever, and permitting a gain of weight.

CONCLUSIONS

This communication draws attention to the value of careful examination of material obtained in the syringe needle when exploring the chests of cases of this type.

Of the cases which are described the chief point of clinical importance is that they demonstrate that even a young infant has a great power of recovery from tuberculous lesions of the lungs, a view which Armand-Delille and others have maintained for some years.

REFERENCES

- ARMAND-DELILLE, P. F., and others. 1927. *Rev. franç. Pédiat.* 3, 1927, p. 1.
ELIASBERG, H., and NEULAND, W. 1920. *Jb. Kinderheilk.* 93, 1920, p. 88.
GÖRTER, E., and LICNAC, G. O. E. 1930. *Acta paediat., Stockholm*, 10, 1930, p. 87.

V. *The Nature of Disease in Infancy*¹

IT might be expected that a study of infantile mortality rates analysed under the certified causes of death would give some idea of the nature and prevalence of diseases in infancy; but those with experience of existing methods of certification know how false is such a hope. During 1939 Dr. F. J. Miller and I conducted a personal inquiry into the 272 infant deaths in Newcastle which revealed the inaccuracies and difficulties of certification. The inquiry included every infant death in the city; in each case the attending doctor was interviewed. When the child died in hospital all records and post-mortem findings were available. The practitioners gave frank and full co-operation. Nearly 10 per cent. of the deaths (25 out of 272) had taken place without the child having received previous medical attention; usually these were sudden or unexpected deaths from brief illnesses, yet an attempt had to be made to certify the cause. It was found that at least a third of the total had been certified with an inaccurate diagnosis. Most doctors admitted their difficulty in reaching a satisfactory diagnosis, but pleaded the necessity of putting down something which would satisfy the registrar. When in doubt some used stereotyped but often irrelevant diagnoses which they knew would be acceptable. These habits were not confined to family practice, for they were found also in hospitals. To give a few examples: a child dead in the third day of a cerebral haemorrhage was certified as inanition and marasmus; another dead of an acute virulent infection on the fourteenth day was certified as atelectasis and prematurity; a child dead of an acute infection 7 days after circumcision was certified as infantile convulsions; a child of 3 months dying after a long struggle against neonatal sepsis was certified as malnutrition and inguinal hernia. On evidence like this deductions about the prevalence of disease, and the construction of plans of prevention, are haphazard undertakings. When, in discussing infant mortality in 1913, Sir George Newman wrote: "The principal operating influence is the ignorance of the mother and the remedy is the educa-

¹ The Bradshaw lecture delivered before the Royal College of Physicians on 7 March 1940. Published in the *Lancet*, 240, 1941, p. 777.

us to classify them as inborn errors of metabolism, but since the disorder is often transient and recovery follows, the two groups of diseases should be differentiated. Icterus gravis neonatorum and anaemia haemolytica neonatorum are regarded by Parsons, Hawksley, and others as temporary failures of the new-born child to regulate its own blood-forming and blood-destroying processes. Haemorrhagic disease of the newborn is probably another example of this type of disease, in spite of the recent suggestion that it is due to a vitamin deficiency.

Defective diet

Whatever defects of diet may be found in older children there are few infants in these days who are not living on an ample and highly vitaminized diet. Scurvy, tetany, and nutritional xerophthalmia are now so rare that most young doctors have never seen them; even rickets is seen only occasionally and in circumstances of frank neglect. But a highly vitaminized diet is not necessarily an ideal diet. In spite of the improvement in artificial feeding of infants and the disappearance of severe forms of nutritional disease, the possible ill effects of bottle-feeding in comparison with breast-feeding have not been accurately determined. Where artificial feeding may not be the immediate cause of death, it is often an accessory before the fact, and no statistical studies of death certificates will reveal this. The study of nutritional disease is turning from the primary forms of dietetic deficiency to the secondary or conditioned forms in which some underlying disease of stomach, bowel, or liver is interfering with absorption or assimilation of food. Before it is too late it should be possible to turn the clinical studies of deficiency disease in yet another direction: by interrogating people who are known to have had severe rickets or scurvy in infancy it should be possible to determine if these conditions have had any remote effect in later life. Brief exposure to lead poisoning in infancy causes chronic nephritis 20-30 years later; perhaps the cause of some of the degenerative diseases of adults may be found by paediatric studies.

NEUROMUSCULAR DISORDERS

The term 'neuromuscular disorders of organ function' was used by John Thompson in discussing the mechanism of pylorospasm

and pyloric stenosis. The clinical effects of those disorders may be present at birth or they may not come into full force until a few weeks after birth. They tend to subside after the sixth month. There is no direct evidence to suggest that they are true congenital anatomical abnormalities. The subsequent history of the affected infants shows that they are not more prone to psychopathic or endocrine disorders than other children. Perhaps the true *causa causans* is a temporary absence for a few months of a chemical substance necessary for co-ordination of the neuromuscular functions. The most evident example is pylorospasm with or without hypertrophy of the pyloric muscle; others are congenital laryngeal stridor, and the various degrees of colonic dysfunction from spastic constipation to Hirschsprung's disease. There is also adynamic ileus of the newborn described by Perrot and Danon (1935), and Gillespie and Rogers (1939). The symptoms of this are evident from birth, and at first are indistinguishable from those due to an impermeable intestinal obstruction; the obstruction, however, is not due to constriction or occlusion but to spastic closure of a segment of the small bowel, the lumen of which is present. In some cases this functional disorder rectifies itself in the second week of life and a spontaneous cure takes place.

CASE 1. A first child of young parents was born at term after a normal pregnancy and labour. At birth it was an apparently healthy male infant, and it cried and slept after birth in a normal manner. On being put to the breast towards the end of the first day there was a little vomiting, and on the second day, when breast-feeding was established, vomiting became more evident. Then for 8 days there was complete obstructive vomiting of deeply bile-stained material. The infant retained his appetite and breast-feeding was continued. I saw him on the sixth day, when the diagnosis of intestinal obstruction due to a congenital atresia of the bowel seemed obvious. Radiographs showed a big stomach with barium passing a short way down the jejunum but not beyond. On the eighth day vomiting was less severe. Thereafter there was a gradual improvement and at 3 weeks all vomiting had disappeared. The child is now a boy of 9 in excellent health.

It may be argued that this was intestinal obstruction due to a local constriction and not to adynamic ileus, but the site of the obstruction and the clinical features so resembled other cases of

adynamic ileus confirmed at operations or necropsy that the diagnosis can be sustained. Gillespie and Rogers in reporting a similar case made their diagnosis after laparotomy. Their patient also recovered.

Other forms of neuromuscular disorder of bowel function of slighter degree are common. The following is, I think, an example, and presents a recognizable clinical entity.

CASE 2. A healthy breast-fed male infant was well during the first 8 weeks of life, advancing steadily in weight from 7 lb. to 9 lb. 4 oz. At 8 weeks he slept and fed with exemplary regularity; his bowels were a little constipated and irregular, but there was nothing obviously abnormal about the appearance or odour of the stools. In the eighth week he began to appear uncomfortable. Sleep was broken and there was slight occasional vomiting; once or twice he vomited unexpectedly in a manner slightly suggestive of pyloric stenosis. At 11 weeks the weight had fallen to 8 lb. 12 oz. On a diagnosis of 'feeding disorder' the mother contemplated weaning the child. I saw him at this time; the breast milk was more than adequate, so breast-feeding was continued; he fed eagerly, but between feeds had periodic discomfort and slight irregular vomiting. The bowels were now irregular; with a suppository or rectal tube a creamy, slightly offensive stool was obtained. The abdomen was prominent and through its thin wall slight distension of the small intestine was visible. There was no evidence of pyloric stenosis, or tuberculosis, or of other infective disease. During the next 10 weeks breast-feeding remained adequate; vomiting subsided but there was still no gain in weight. The main symptom continued to be irregularity of the bowels, with some abdominal distension. At the age of 4 months he was still under 9 lb. in weight, sleeping irregularly and passing his waking hours in alternating phases of alert interest and peevish discontent. With no other treatment than the use of a rectal tube or suppository, improvement set in in the fifth month. Bowel movements became more regular; there was a steady gain in weight and complete recovery followed. Throughout the illness the child remained breast-fed, attended only by its mother and free from any contaminating infection. Occasional gastric lavage and a little Eumydrin were used in the early weeks of the illness, but without any effect.

In the past this might have been described as a case of fat-intolerance, carbohydrate dyspepsia, protein damage, or abortive coeliac disease. I am reasonably sure that the fault was a neuromuscular disorder of the small bowel interfering with normal function but never sufficient to produce obstruction or visible peristalsis. In adults a single simple narrowing of the small intestine may produce a disease picture resembling sprue; there are therefore grounds for assuming that a disorder of peristaltic action in the bowels of an infant may cause an apparent feeding disorder with wasting—indeed, some cases of coeliac disease itself may be due to disturbances of this nature.

Another possible manifestation of neuromuscular disorder of function is to be seen in apparent respiratory obstruction. In some infants the obstruction appears to be in the nasopharynx, in others in the bronchial tubes. To the observer the children give the impression that they have not yet learnt to breathe normally. The disability may produce the deformity of pigeon-chest. After a few months most of them begin to recover from their disability; those who die in the meantime from intercurrent disease are found to have no obstruction in the respiratory tract. I can find no other explanation for them than neuromuscular disorder of the respiratory mechanism; and there is evidence that such disorders of function are commoner in infancy than is supposed.

INFECTIVE DISEASES

In a town with an infantile mortality rate of 60 per 1,000 live births it is probable that at least half the infant deaths are due to infective or bacterial diseases. With higher mortality rates the proportion of deaths due to infection will be still greater. This conclusion is based on our Newcastle inquiry. Most infective illnesses could be prevented, and therein lies the reasonable hope that an infantile mortality rate under 30 per 1,000 live births should be attainable.

In older children and adults the recognition and classification of infective diseases on an aetiological basis is relatively easy. In infants not only may the causative organism be difficult to recover but the pattern of the illness following any known infection is so variable as to give little help in diagnosis. A *Bacillus dysenteriae* Flexner infection may cause acute generalized toxæmia in one

infant, meningitis in another, and dysentery in another; two infants may be similarly infected and both may die, yet in one the illness may have been febrile and in the other afebrile. Some infective illnesses will kill an infant within 24 hours, thus allowing little time for the gathering of information. An organism such as the *B. coli*, which is relatively innocuous to older children, may cause a rapidly fatal illness in a new-born infant, and an investigating bacteriologist with fixed ideas of what are pathogenic organisms may be led astray.

It is difficult to estimate the prevalence in infancy of the infective diseases which can be traced to known organisms. Cameron (1929) and Ogilvie (1933) have described the dangers of sepsis neonatorum. MacGregor (1939) has pointed out the prevalence of neonatal pneumonia as a cause of death shortly after birth, and Craig (1936) and others have shown how the *B. coli* and other coliform organisms cause meningitis in the newly born. Cass (1940) has outlined the effects of the staphylococcal diseases. In America Rice, Best, Frant, Abramson (1937), and others have made similar observations. These give no clue to the relative incidence of the various infections in infancy. Our Newcastle inquiry was carried out in a year free from serious epidemics and at a time when institutional infections of infants were well controlled; there were about a hundred deaths from infective diseases of which 6 were due to tuberculosis, 1 to diphtheria, 1 to syphilis, and 9 to whooping-cough; there were 12 deaths from illness which could be called acute infective gastro-enteritis; the remainder died from acute infective illnesses, some of which appeared to be acute respiratory infections, but the majority presented no localizing symptoms on which to base a diagnosis.

To study and prevent these acute infections in infancy is one of the chief duties of paediatrics, for many pass undiagnosed and are not investigated. Most of us are familiar with the infective nature of a disease with outward signs like pemphigus neonatorum, but there are others with no localized signs which may kill within 24 hours or lead to wasting and feeding disorders. The variety of pathogenic organisms is great, but they all tend to produce septicaemia with symptoms of drowsiness and a disinclination for food. This may be present within 24 hours of birth with localization of the infection as a neonatal pneumonia; or more typically an infant may thrive well for a few days, then refuse feeds, become drowsy or

peevish, develop a little diarrhoea, and then recover or die. At the height of the illness some show an interesting physical sign which I call 'canine breathing'; a short panting form of respiration rising to a rate of 120 to 130 a minute, but not resembling pneumonic breathing. The clinical peculiarities of neonatal infections are set out in a report by Guthrie and Montgomery (1939) of illnesses in a Glasgow hospital due to the *B. enteritidis* of Gaertner in which the infants had no dysenteric symptoms but only signs of mild intestinal catarrh followed by septicaemia, meningitis, or cholecystitis. A new-born infant who becomes abnormally drowsy and refuses its feeds and is not suffering from cerebral haemorrhage should be suspected of an acute neonatal infection.

In later infancy—that is, at any time after the neonatal period—the acute infections may assume forms lasting from a few hours to several weeks, and ranging from fatal toxæmia to mild malaise. A description of these, although explaining nothing, may serve a purpose.

The first is an acute illness lasting less than 48 hours with no localizing signs and ending in death or recovery. The infant is prostrated or comatose—a state described in the northern vernacular as 'dead felled'. The illness may be so short as to have run its course before medical aid is summoned, and into this category must be put some of the cases of infants found unexpectedly dead in bed.

CASE 3. A healthy child of 7 months in good social circumstances was noticed one afternoon to have an almost insignificant nasal catarrh. Next morning she was kept indoors but played cheerfully in her nursery. At 3 o'clock in the afternoon she was feverish and desired to be nursed; by 5 o'clock she was limp and quiet. At 7 o'clock she was drowsy and refused drinks. Seen between 8 and 9 o'clock the same evening the child was semiconscious but inclined to throw herself about. There were no localizing signs. Urine: no albumin and no pus cells; not cultured for organisms. The cerebrospinal fluid was clear and normal. The child became comatose, and after a minor convulsion died at 4 o'clock in the morning. The duration of the acute illness was thus a little less than 12 hours. A post-mortem examination was made and no anatomical lesions sufficient to explain death were found. Cultures from the heart-blood were not made.

These fulminating forms of infective illness are not uncommon

in infants, and have often been described as due to acute respiratory infections with streptococci. It is clear that many organisms other than the streptococcus may be the cause, and there may be no clinical or pathological evidence of respiratory disease. I have seen many illnesses of this type in which the determined co-operation of clinician and pathologist has failed to reveal the cause of the septicaemia.

A second type of infective illness is a symptomless pyrexia lasting 4-10 days and ending usually in complete recovery. Starting insidiously or acutely, there is feverish drowsiness, with disinclination for food or occasional sickness, but with no localized symptoms or signs. The condition may pass to toxæmia with dehydration. A trace of albumin with a few leucocytes may be found in the urine, and in this and other ways the picture resembles a paratyphoid infection. Engorgement of the fauces may suggest that site as the *portal of entry of infection*. This type of illness is to be seen occasionally after vaccination or circumcision, even when the primary lesions show no signs of septic infection. Recovery is usually uneventful.

A third type of infective illness starts suddenly or insidiously as an acute symptomless pyrexia and after a few days localizes to produce signs of organic disease—particularly pneumonia, otitis media, pyelocystitis, gastro-enteritis, meningitis, or skin sepsis.

A fourth type is acute primary gastro-enteritis, the 'cholera infantum' of former times. There is a rapid onset of symptoms with vomiting; the stools become dysenteric or copious, frequent, and loose. The characteristic facies, with grey pallor and dehydration, is quickly established; mortality is high. This type of illness can probably be caused by many organisms. In some outbreaks dysenteric organisms are found, but in newly born or ailing infants organisms which are apparently benign to adults may be the cause.

A fifth type of disease is one in which there is no invasive fever or septicaemia and the infection reveals itself first by a localized lesion or multiple localized lesions without symptoms of a generalized illness. These may appear in the skin, producing onychia and abscesses, or in deeper sites producing osteitis, arthritis, otitis, empyema, or indeed any local lesion. This type is characterized by pus formations, and brings to light an important principle of infective disease in infancy. If an infective disease becomes localized with pus formation an infant shows more power of local resistance

than an older patient, provided the infection is not secondarily contaminated by other organisms. This explains the unusual power of recovery which some infants show from staphylococcal osteitis and empyema with no treatment other than occasional aspiration of the pus. Such cases have been reported by Cass (1940), and the following is an example of one of them.

CASE 4. A healthy male child began to develop swelling just above the left knee-joint at the age of 2 weeks. This increased steadily until, at 4 weeks, there was obvious reddening of the skin with swelling of the knee-joint and lower end of the femur. The child's general condition was not disturbed; he took his feeds well throughout and continued to gain weight. The left sternoclavicular joint became similarly swollen, red, and tender. The knee-joint was aspirated four times in 10 days; on each occasion the pus contained *Staphylococcus aureus*. A radiograph of the femur showed definite osteitis, the course of which was followed by further radiological examinations. No operation was performed; the osteitis and arthritis in both joints subsided within 3 months and a spontaneous cure took place, the child remaining well a year after.

A description of the clinical course of acute infective disease does nothing to explain by what process an infant may be killed within 24 hours with no local evidence of the primary site of infection, or why organisms like the dysentery bacillus have such a tissue specificity in the older child but lack it in the infant. It does not explain by what means an organism like the *B. lactis aerogenes* may be fatally pathogenic to an infant, to become quite innocuous in later life. I have two outstanding impressions of infective illnesses in newly born and young infants; first, the sudden explosive character of the septicaemic illness without local evidence of the primary site of infection; and second, the frequent absence of any septicaemic or constitutional symptoms in those few infants who can oppose the infection by immobilizing it in a local purulent lesion. Rich (1932) experimenting with rabbits showed that inflammation is not a barrier of resistance laid down to oppose a spread of infection but a local phenomenon at the site of infection in tissues which have been sensitized by a previous infection with the same organism. This phenomenon is well known in the case of tuberculous infection, and may be true of most of the organisms which later live a harmless saprophytic life in selected tissues of the adult body.

The infant may have to pass through a stage of adjustment after primary infection with each of these organisms, and the primary infection may take place without evident clinical disease arising; or it may take place in a manner and at an age when the response will be different from that provoked by reinfection in later life. We may have to study the primary or infantile forms of all infections, just as we have studied the illnesses produced by primary infection with tuberculosis. This branch of clinical medicine offers unusual opportunities for research and discovery. So far in England this work has been done mainly by amateurs—using the word in the sense of those who do something for the love of it. But now the importance of the subject demands that those with suitable talent and training shall be selected to do the work in places suitable for research. The training implies a clinical experience of paediatrics. The places imply a staff and equipment which will ensure the safe custody of young infants.

REFERENCES

- CAMERON, H. C. 1929. *Lancet*, 216, 1929, p. 1127.
 CASS, J. 1940. *Arch. Dis. Childh.* 15, 1940, pp. 55 and 85.
 CRAIG, W. S. 1936. *Ibid.* 11, 1936, p. 171.
 GILLESPIE, J. B., and ROGERS, J. C. T. 1939. *Arch. Pediat.* 56, 1939, p. 269.
 GUTHRIE, K. J., and MONTGOMERY, G. L. 1939. *J. Path. Bact.* 49, 1939, p. 393.
 MACGREGOR, A. R. 1939. *Arch. Dis. Childh.* 14, 1939, p. 323.
 OGILVIE, A. G. 1933. *Ibid.* 8, 1933, p. 413.
 PERROT, A., and DANON, L. 1935. *Ann. Anat. path. méd.-chir.* 12, 1935, p. 157.
 RICE, J. L., and others. 1937. *J. Amer. med. Ass.* 109, 1937, p. 475.
 RICH, A. R. 1932. *Bull. Johns Hopk. Hosp.* 52, 1932, p. 203.

VI. *Pink Disease*¹

DEFINITION

(*Synonyms.* Swift's disease; acrodynia; erythroedema; Feer's disease)

THIS is a disease causing inexplicable misery, photophobia, sleeplessness, hypotonia, and wasting in young children which may be difficult to diagnose unless it results in the red, sodden appearance of hands and feet from which it derives its name of pink disease.

HISTORY

Swift (1914) and his Australian colleagues were responsible for the first complete clinical studies of pink disease, and although the peculiar nature of the malady had aroused the attention of earlier observers, his name deserves to be given to the disease if an eponymous title is to be used. In 1903 Selter reported 8 cases of what may have been pink disease, naming it 'trophodermatoneurosis', but the published account of his cases is so fragmentary and indefinite that little can be made of them. Quite independently Bilderback (1925) recognized the disease in America, and Byfield (1920) described it as 'a polyneuritic syndrome resembling pellagra-acrodynia'. Simultaneously Feer (1923) of Zurich was observing and recording the disease as a 'peculiar neurosis of the vegetative nervous system in young children'. The disease was first reported in England by Thursfield and Paterson in 1922, and since then it has been widely recognized in all parts of the world.

AETIOLOGY

The cause of pink disease is not known. It affects both male and female children. The first symptoms usually arise between the ages of 3 and 9 months. The accompanying chart (see Fig. 3) represents a series of cases personally observed in a period of 20 years in

¹ From *British Encyclopaedia of Medical Practice*, 2nd ed., London, Butterworth, 1950-2, 9, p. 609.

which the youngest was 8 weeks and the oldest 2 years and 5 months at the beginning of their illness. I have seen only 1 case outside this age period, a boy of 4 years briefly demonstrated to me by Moro at Heidelberg many years ago. Unless there are geographical variations in its age incidence which carry it beyond the

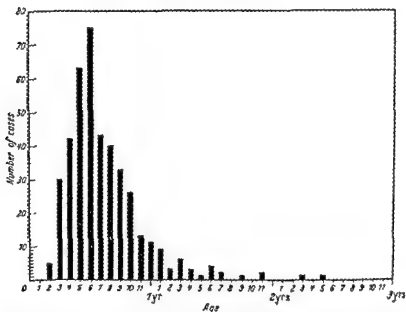


FIG. 3. Age incidence of onset of pink disease at the Babies' Hospital and the Children's Clinic, Newcastle upon Tyne—the youngest at 8 weeks, the eldest at 2 years and 5 months.

range of my experience I shall doubt the diagnosis of pink disease in any child over 3 years of age. Some of the reported cases in older children require critical confirmation before they are accepted as pink disease.

Pink disease occurs as readily in breast-fed as in bottle-fed infants, and there is no evidence to support the suggestion that it is a disease of defective nutrition; indeed, many observers have remarked that it is more likely to occur in well-cared-for children than in those from poverty-stricken homes. In so many instances the infant's diet has been reinforced by orange juice, cod-liver oil, Marmite, and other foods rich in vitamins that the nutritional

cause of the disease can be discarded. Significant variations in its geographical distribution have been observed, and it appears to be unduly prevalent in the neighbourhood of certain towns in northern England.

Pink disease is not contagious, but it has been noted that successive infants in a family may be attacked. It has been known to

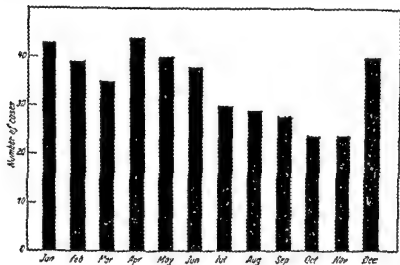


FIG. 4. Monthly distribution of 404 cases of pink disease.

affect one twin child and not the other when both have been similarly fed and in daily contact with each other. It has occurred simultaneously in identical twins.

It has been suggested that pink disease occurs most frequently in the spring or summer months, and that an abnormal reaction to sunlight plays some part in its causation. Wood and others have refuted this hypothesis of light hypersensitivity. Fig. 4 represents 404 patients in whom the months of onset of the disease were exactly determined. This does not reveal any significant variation of its incidence in the north of England.

Another hypothesis of causation is that it is due to a particular virus infection, or to a common viscerotoxic virus which in susceptible children occasionally takes on a neurotoxic role to affect the sensory nerve endings.

reveals the nature and the torture of the discomfort. The child frowns constantly or assumes a look of mute appeal. Weary but unable to sleep, tired but incapable of resting, it looks up for sympathy and turns away in disgust as if aware that the onlookers are incapable either of understanding its sufferings or of relieving its distress. Such sleep as the child can snatch is usually taken with its face pressed into the pillow, which no doubt is a means of temporarily alleviating the circumoral irritation. The hands and feet are now swollen and look pink, sodden, or cyanosed. The nose and cheeks may be affected in the same manner. The skin of the trunk is moist and covered with a fine sudaminous rash and then becomes streaked with scratch marks. At this stage there is a tendency for deep ulcerating lesions to develop in the swollen fingers and in the mouth. From their appearance these suggest trophic sores, particularly in the gum margins, which may become detached and carry with them loosened teeth. Even the tongue may be affected, and it is reported (Spence, 1932) that a boy with pink disease chewed off the end of his tongue in an attempt to relieve his agony. The pulse is rapid and may reach 200 a minute. The blood-pressure is raised.

The child remains beset by these distressing symptoms for 2 or 3 months and then passes into a stage of slow recovery, unless complications intervene. The stage of recovery is ushered in by the return of a smile. The child begins to take an interest in his toys. His appetite returns, and muscular strength and weight are slowly regained. The whole course of the disease is usually 3 or 4 months. It may be shorter, but a few children may be ill for as long as 12 months. The hypotonia is the last symptom to disappear.

Skin sepsis, ulcerative lesions in the mouth, and broncho-pneumonia are the commonest complications. Broncho-pneumonia has a special significance, for it may lead to a persistent loose cough, and occasionally after many years bronchiectasis results. Two alimentary complications not infrequently occur and may endanger life: intussusception and prolapse of the rectum.

Some observers have drawn attention to the loss of knee-jerks as a sign of pink disease and suggest that this is due to a peripheral neuritis. This is probably a faulty observation, for the picture is not that of a peripheral neuritis, and in many patients the knee-jerks are retained and active.

Another symptom or complication which deserves attention is

the tendency to a sudden and unexpected death suggesting acute heart failure. This may be presaged by a rapid change in which the child ceases to wail and writhe and lies ominously quiet and anxiously awake with a pulse-rate rising beyond 200 a minute for a day or two before death.

There are probably both abortive and masked forms of pink disease. In the abortive forms the symptoms subside after a few weeks of misery and photophobia, and before severe constitutional disturbance has arisen. In these circumstances diagnosis is difficult. It is difficult also when the disease is masked by severe intercurrent illness, such as broncho-pneumonia, for in the presence of a complication of this sort the typical writhing and discoloration of the hands may vanish. If the child is seen for the first time in that state, the diagnosis must be attempted on a description of the illness which preceded the acute complication.

A final warning about diagnosis may not be out of place. There is an increasing tendency to over-diagnose pink disease where misery is present. The best technique is to watch the behaviour of the child on his mother's knee and to do this if necessary on many occasions.

PROGNOSIS

In the absence of complications the mortality is low. Wood and Wood (1935) have pointed out that recovery depends on the care and nursing given to the patient in the acute stage. In those treated at home, where constant attention was given, the mortality was 3 per cent., whereas in those treated in hospitals, where ward infections were frequent, the mortality was 30 per cent.

A measure of the severity of the illness and prospect of recovery is revealed in the loss of weight. In a very severe case a child of no more than 20 lb. in weight may lose 4 or 5 lb. The outcome of the illness, however, depends less on this than on the avoidance of broncho-pneumonia, a dire consequence always to be feared if oral sepsis arises to provoke it.

TREATMENT

There is no specific remedy for pink disease. The best that can be done is to prevent complications, and, after explaining to the

VII. *Poliomyelitis*¹

POLIOMYELITIS is the disease caused by one or other of the poliomyelitis group of viruses influenced by distinctive aetiological factors such as age and season, and resulting in paralysis or in other illnesses indicating disease in the central nervous system; but which may result also in abortive or subclinical illnesses difficult to diagnose on clinical evidence alone. If this definition appears too cautious it should be remembered that our concept of the aetiology, the epidemiology, and the clinical picture of the disease has altered greatly during the past 50 years, and is still altering in such ways as to make a more precise definition risky.

The present trends are: (1) in pathogenesis to get a better understanding of the various strains of the virus and the manner in which they invade the body, multiply in its tissues, damage the cells of the nervous system, or yield to the body's resistant forces; (2) in aetiology to explain the rise in age incidence of recent years, and the part played by muscle injury or exhaustion in precipitating paralysis; and (3) towards a closer study of the clinical picture by more precise and continuous methods of observation in order to identify the abortive and non-paralytic illnesses of the disease.

In broad outline the natural history of poliomyelitis should be looked at from the point of view both of the virus and the host. Until about 50 years ago there was an equilibrium between them. The virus was then ubiquitous within a community to the extent that most young children would be infected with it before the age of 3 years. This initial primary infection was probably spread by faecal contaminations and digital contacts which were inescapable in the households of that period, or perhaps by food and oral contacts. Although primary infection in infancy was universal, only rarely did it lead to a paralytic illness. Such was the equilibrium between virus and host. The disease, if it occurred at all, was then truly infantile paralysis and affected young children only. The older children and adults lived immune to the virus of their community. If an older child or an adult was infected it indicated either

¹ From *Modern Trends in Paediatrics*, edited by L. G. Parsons, London, Butterworth, 1951, p. 298.

that he had unhappily escaped infection in infancy, or that he was overcome by a foreign virus, a stranger within the gates, so to speak.

During the past 50 years the equilibrium has been broken by three new forces. The first is the new domestic hygiene which diminishes the chance of an immunizing infection in infancy. The second is the restriction of family size which diminishes the chance of intra-familial infection between children. The third is the ease of travel which allows the quick transfer of strange viruses from one community to another.

Under these new social conditions and altered biological environments the virus has increased its power to invade the central nervous system. Originally a restricted viscerotropic virus, it is now neurotropic. And the more 'virgin soil' there is in older children, in proportion to the decreasing number infected and immunized in early infancy, the more neurotropic it becomes. The chance of paralytic illness or death following primary infection increases with the age of the patient. Such is the trend of poliomyelitis as seen in the light of its natural history.

HISTORY

A knowledge of the history of poliomyelitis throws light both on the changing concepts of the disease and on the changes in its epidemic constitution.

Romer, whose monograph in 1913 was a landmark, took the heroic view of history when he wrote: 'We owe our knowledge of this disease to a few exceptional men. Others have done much in a small way to fill the gaps left by the men of wide views and imagination, but as time progresses their work will become more and more of an anonymous character.' He had in mind Heine, Medin, Wickmann, and possibly Landsteiner. Names of other exceptional men could be added, whose experimental work in recent years has revealed the pathogenesis of the disease and the multiplicity of its clinical effects. But Heine, Medin, Wickmann, and Landsteiner are the early stars of this firmament.

Jacob Heine (1800-79) wrote the first classical description of the disease in 1840 and gave it the name 'spinal infantile paralysis'. He was a forthright man who rejoiced in overcoming difficulties, which he did by entering school first at the age of 21 years to gain admission to a university at 23 years. At the age of 30 years he

took charge of a cripples' home. Out of the medley of its patients he isolated a group the clinical history and residual paralysis of which set it apart as the clinical entity we now know. His description remained for years the basis of the accounts in all the textbooks, and no important addition was made to it until Medin (1890) and Wickmann (1907) studied poliomyelitis in its epidemic forms in Sweden and said that it was a symptom complex which might appear also in a bulbar, central, polyneuritic, ataxic, meningitic, or abortive form, or as an ascending paralysis which had hitherto been described by Landry. In the meantime French neurologists had been busy studying its histology which was important, and its muscle reactions which were not important; and Charles West in 1852 added a note on the unexpected nature of the disease by calling it 'morning paralysis'.

Heine had called the disease 'spinal infantile paralysis'. Later, when histological studies defined the lesion, the name 'acute anterior poliomyelitis' was conceived. The epidemiological studies in the eighties brought forward the names 'epidemic infantile paralysis' and 'epidemic poliomyelitis'. The insufficiency of this terminology caused Wickmann to propose the name 'Heine-Medin disease'.

In the 'era of the cocci', experimental search for a bacteriological cause of poliomyelitis gave only negative results, but in 1908 Landsteiner and Popper published their brilliant work in which they transmitted the disease to monkeys by intra-peritoneal injections, using as infective material the spinal cord of a child of 9 years of age who had died on the fifth day of the illness. This established the virus theory of the causation of poliomyelitis. The experimental work in monkeys was advanced by Romer (1909, 1910, 1913) and by Flexner and Lewis (1909) using new methods of experimental infection, and finally placed on a firm basis by the Swedish workers Kling, Pettersson, and Wernstedt (1912) who were impelled by their experience of the epidemic of 1911. From that point work forged ahead in the clinical, epidemiological, and experimental fields. We can do no more than mention Aycock, Horstmann, Burnet, Bodian, Howe, Paul, and Sabin among the host of others who have done so much in this difficult but intriguing subject.

Amongst the important contributions to this knowledge have been the studies of the epidemiology of poliomyelitis starting with Wickmann's observation on the changing epidemic constitution of

the disease in Sweden, the first good critical summary of which appeared in the report of Kling, Pettersson, and Wernstedt to the International Congress of Hygiene in 1912.

THE VIRUSES OF POLIOMYELITIS

Until a few years ago it was believed that poliomyelitis virus occurred naturally in man only, and could be transmitted to monkeys and apes only. Now it is known that some strains of virus can be transmitted also to rodents and may occur naturally in them and in swine and mice.

In the early work of the Landsteiner-Flexner period the transmission of the noxious agent through a series of monkeys proved it to be a living virus and not a lifeless poison. It was proved to be a filter-passing agent that could be preserved in glycerin. The virus was at first recovered only from the brain and spinal cord, but Flexner demonstrated it later in the mucous membrane of the nose, while he failed to find it in the secretions from the mouth. That was in the early days of experimental work and now the virus has been recovered from nasopharyngeal washings, from tracheal secretions, from pharyngeal secretions, from the pharyngeal and intestinal walls, from faeces, from mesenteric and cervical lymph nodes, but less easily from cerebrospinal fluid and from blood, and not from urine. It has been recovered from healthy carriers and from patients in all active stages of the disease both in its non-paralytic and paralytic forms.

The discovery of virus in the stools of pre-paralytic and paralytic patients, and in apparently healthy contacts with these patients, and its presence in the sewage of towns in epidemic periods (Paul, Trask, and Culotta, 1939) has opened an entirely new aspect on the epidemiology of the disease.

Burnet (1948) describes the virus as extremely small and calculated to have a diameter of 10 $m\mu$, 1 $m\mu$ being one-millionth of a millimetre. There is some evidence that its shape is that of a slender fibre. The virus is relatively stable and can survive for considerable periods outside the body, for example in faeces. It is incapable of multiplication in any other environment than the interior of a living susceptible cell.

Most strains of virus from the human disease are transmissible to monkeys and cannot be adapted to rodents. These are spoken of

as 'monkey pathogenic'. Other strains can be transmitted to rodents and are called 'rodent pathogenic'. The Lansing strain, isolated by Armstrong, is one of these. When Theiler in 1934 discovered a strain occurring naturally in mice, yet another field of experimental work was opened up. For a discussion of the many strains that have been isolated the reader is referred to the article by Rhodes (1947). It only remains to be noted here that antigenic differences have been observed between different strains but that some tend to fall into groups with antigenic interrelationships. Three broad groups of virus strains have been defined: (1) the monkey pathogenic strains, (2) Lansing, S.K., and M.M. strains, and (3) Theiler's mouse strains. The modes of transmission, excretion, and survival of the virus will be discussed under pathogenesis and epidemiology.

The discovery by Dalldorf and Sickles (1948, 1949) of the Coxsackie viruses, so named from the village in New York State where they were first isolated, has a bearing on poliomyelitis, if only because they cause illnesses with painful myalgia, aseptic meningitis, and paralysis difficult to distinguish from poliomyelitis. The discovery illustrates both the trend and the need to correlate clinical observation with virus studies in laboratories.

AETIOLOGY

The aetiology, the pathogenesis, and the epidemiology of poliomyelitis have such close bearing on each other that it is difficult to know which to describe first in an attempt at clarity. But we shall proceed in the order mentioned in an attempt to indicate the trend of the disease, before describing its clinical features.

The main aetiological factors to be considered are the age of attack, race, social grouping, the seasonal distribution, and the effect of injury or fatigue in precipitating the disease.

Age

As the original name of the disease testifies, it was at first considered to occur only in young children under the age of 3 years. *This may have been true at the time when Heine first discovered the disease*, but during the past 50 years poliomyelitis has broken loose from that limitation, and as time goes on older age-groups tend to become affected. The facts on which this statement is based are clearly substantiated in the precise epidemiological

studies of recent years. For example, in Denmark in 1911-14 only 10 per cent. of the patients were over the age of 15 years; in 1934 there were 31 per cent.; and in 1944 there were 53 per cent. over that age. This conforms to the findings in America, Australia, Scandinavia, and in Great Britain, as was demonstrated in the epidemic of 1947.

This changed incidence has been most apparent in the more civilized countries where improved hygienic techniques diminish the spread of universal infections in infancy, and render a larger proportion of the population vulnerable at an older age. In spite of these changes it remains true that the younger are the more prone to be infected and diseased, but the fact may be obscured by a state of affairs in which the younger the child the less likely is it to develop a paralytic and therefore recognizable illness. When epidemics make their first appearance in countries where the disease has been infrequent, the majority of the paralytic illnesses are likely to occur in children under the age of 5 years. In the Southern States of America, Wenner (1946) found that children under 5 years of age were affected three times more often than children aged 5-10 years, whereas in Connecticut at the same time children aged 5-10 years were affected as frequently as those under 5 years. In the 1947 epidemic the age distributions in England and Wales are shown in Table 3.

TABLE 3

Age distribution of poliomyelitis, England and Wales

<i>Age in years</i>	<i>Notifications</i>	<i>Percentage</i>
0-4	2,391	31.5
5-9	1,610	21.2
10-14	1,112	14.7
15-24	1,218	16.1
25 and over	1,254	16.5

The limits of age incidence are very wide. The disease has been reported in new-born children and in adults past the age of 50 years.

Sex

Sex plays little part in aetiology. The nearest we can get to facts is that the International Committee (1932) summarized the data of

some 36,000 cases and found the male:female rates were 1·3:1. Sex has, however, an influence on mortality. Poliomyelitis kills more males than females.

Race

Judged by the incidence of paralytic illnesses and the size of epidemics, some races are apparently more affected by poliomyelitis than others. In North America its incidence in the whites is 3-4 times greater than in the negroes. The movement of soldiers during the Second World War is an experiment with interesting results. In general, when white soldiers live amongst primitive communities, as did the occupational forces in Japan, they are more prone to paralytic poliomyelitis than is a similar group at home, and much more prone to get poliomyelitis than are the natives. It can be assumed that poliomyelitis occurs in all parts of the world and that the difference in racial susceptibility is to be explained on immunological grounds and not on inherent racial differences.

Social factors

While sporadic poliomyelitis and small outbreaks are widespread, big epidemics have arisen in those countries with the highest standards of social life. Nevertheless no social class is entirely immune as no people and no country is immune. Whereas soldiers in India show a higher incidence than in home bases, the incidence amongst officers was about five times greater than in the non-commissioned ranks. These apparent social differences may also be explained on grounds of immunology by which the higher social classes, in separating and segregating their children, thereby create a more susceptible constitution in their social class.

There appears, however, an aetiological relationship between age incidence and social class. A high outbreak rate amongst children under 5 years of age is associated with poor social hygiene. A high outbreak rate in older children and adults indicates either a better social hygiene or the invasion of the community by a new strain of virus against which no immunity had been developed in childhood.

Seasonal incidence

When the disease occurs only sporadically a seasonal incidence

is not striking, but in epidemic form it is a disease of the hot seasons. The 1947 epidemic in Great Britain took place in one of the country's hottest summers. While it lasted it was predicted the epidemic would cease with the advent of cold weather, and it did disappear dramatically in the first week of October. In the Southern hemisphere it occurs predominantly in the first 4 months of the year. Whether in arctic or in tropical climates it occurs in seasonal waves in all places. Gard (1938) studied meteorological influences in Swedish outbreaks and suggested that rainfall might play its part, but nothing conclusive was proved. While we have accepted the summer incidence of epidemic poliomyelitis as one of its most consistent features, even this rule is proved by exceptions. A recent small epidemic amongst Eskimos took place in their winter, and while this article is being written it is reported from Adelaide that their 1949-50 epidemic is continuing into their winter. But by and large we must accept the view that in the temperate countries in the Northern hemisphere poliomyelitis occurs in epidemic form only in the summer with its epidemic peaks in the months of August and September.

Draper's view that the constitutional type of the patient has an aetiological effect in paralytic poliomyelitis is slipping into obscurity. It is so difficult to know what is meant by constitutional type, and since 1917, when he first advanced the theory, no concrete proof has been brought forward to confirm it.

Muscle injury and fatigue

A more hopeful aetiological study is the influence of fatigue and injury in predisposing to the disease. This will be mentioned again under pathogenesis. It is sufficient here to state that the classical observation of Francis and his colleagues (1942) on the 5 children in one family who developed paralytic poliomyelitis after tonsillectomy needs to be studied alongside the effect of pregnancy, fatigue, trauma, and the injury to muscles made by previous injections. I myself have seen sufficiently close correlation between excessive physical exertion in children and the advent of paralytic poliomyelitis to support the view. The facts and arguments about muscle exhaustion and paralysis are well stated by Russell (1949) from his studies of the 1947 outbreak in Britain and earlier by Levinson, Milzer, and Lewin (1945).

If an aetiological association is established between muscle

trauma and paralysis a new field of interest opens both in the pathogenesis and in the prevention of the disease. The accumulating evidence appears to leave no doubt about the association. On the other hand, there are many cases of paralytic poliomyelitis without any history of a preceding trauma or exhaustion.

The alarming report of 5 children in a family with bulbar paralysis after tonsillectomy is one of the outstanding records. The sixth child, not operated on, remained healthy but excreted virus in the stool. Another example of a precipitating trauma is the injury of muscles by injections for immunization. Martin (1950) collected seventeen such instances and his findings have been confirmed by McClosky (1950) and Geffen (1950). The statistical validity of the findings has been established by Bradford Hill and Knowelden. The conclusive part of the evidence is that whereas paralysis from poliomyelitis is two or three times commoner in the legs than in the arms, the paralysis in inoculated children is commoner in the arms than in the legs, and most common in the inoculated arm.

There are many reports of other forms of strain or injury, such as surgical operation and fracture, precipitating a severe or fatal paralysis. Pregnancy also is considered as a possible aetiological factor. If these views are proved, they will confirm the experimental work on the effect of fatigue and cold in increasing the paralysis in monkeys infected with the virus. The practical application of this new knowledge about the effect of injury, fatigue, and cold in poliomyelitis will be obvious. At a time when doctors are so ready to inject penicillin in undiagnosed febrile illnesses, the possible effects of this form of muscle injury should be borne in mind.

The effect of poliomyelitis on pregnancy and vice versa has been discussed by Aycock (1941), Maxwell and Willcox (1947), and others. Biermann and Piszczek (1944) have recorded a remarkable case of a woman, healthy in her pregnancy, whose illness of poliomyelitis started on the first day after delivery. She died with respiratory paralysis on the fourth day. After birth her infant was in contact with its mother for 10 minutes only on its first day of life. It remained well until the eleventh day, and then began its illness of poliomyelitis with paralysis of limbs and abdominal muscles. Baskin, Soule, and Mills (1950) report the birth of a baby to a woman in a respirator critically ill with poliomyelitis of 4 days' duration. When 3 days old the baby developed a temperature,

2 days later some paresis developed, the protein in the cerebrospinal fluid was 300 mg. per cent., and the lymphocytes were 97 per cubic mm.; death occurred on the seventh day of life. Changes characteristic of poliomyelitis were found in the cord, midbrain, and other regions. There are many observations showing that a woman may have paralytic poliomyelitis during pregnancy, and proceed to full term and give birth to an infant who remains healthy.

PATHOGENESIS

We owe much to those experimental scientists who have shown how the virus of poliomyelitis enters the human body and proceeds in its various ways to kill some, to injure others, and yet allow the majority to escape unaffected. The facts are not yet fully known, but the rate of this scientific progress suggests that we may reach a complete understanding of the disease and find means to prevent or overcome it. The number of research workers who have contributed is so great that merely to mention their names would be encyclopaedic, but the articles by Rhodes (1947), and Horstmann (1948), the papers of Faber and Silverberg (1946), Howe and Bodian (1942), Sabin (1941), Burnet, Jackson, and Robertson (1939), and Burnet (1945) will direct the reader.

The virus enters the body through the mouth or nose, either airborne or by ingestion of contaminated food.

It lodges and multiplies in the walls of the air passages and alimentary canal, and passes readily along the nerves to the central nervous system. Its selective sites for multiplication are the pharynx and the small intestine. The physical mechanism of spread is not explained. As Burnet says: 'it poses a number of intriguing unsolved problems'. Reaching the central nervous system multiplication of virus continues, but is restricted to certain cells—the anterior horn cells, the proprioceptive neurones of cranial and spinal ganglia, and their relay in the vestibular nuclei, the roof nuclei of the cerebellum, and the reticular formation on the floor of the cerebellum.

Multiplication and proliferation of virus within the nerve-cells does not necessarily imply either paralytic illnesses or indeed any recognizable clinical illness—for Bodian and Howe (1945) have been impressed with the widespread silent lesions in the brain and

cord without any associated clinical evidence. When paralysis does occur, through irreversible damage of the nerve-cells, multiplication of the virus quickly diminishes.

We might summarize by saying that the virus of poliomyelitis is neurocytophilic, affects efferent neurones concerned in exercise and posture, and can kill the cell, die within the cell, or be present for some time awaiting suitable conditions for multiplying.

Accepting the view that a large number of people are infected with the virus, but that clinical illness or paralysis follows only in a small proportion, we must ask how long these patients and carriers harbour or excrete the virus. The excreted virus would have to originate either from the pharynx or the bowel. Howe, Bodian, and Wenner (1945) showed that virus disappeared from pharyngeal swabs within the first 5 days of the illness. Horstmann, Ward, and Melnick (1944) showed that virus might persist in stools for 8 weeks, but disappear in 50 per cent. by the third or fourth week.

At this point the effect of muscular fatigue or muscle injury on the multiplication of virus in the associated anterior horn cells can be considered. It is known that small physical changes may localize proliferation of the virus. It is tempting to speculate that muscle fatigue or injury has an effect on the associated anterior horn cells that encourages the multiplication of virus within these cells. Tonsillectomy will predispose to bulbar paralysis, injections into the deltoid to shoulder paralysis, exhaustion (by running) to leg paralysis, rowing and horse riding to trunk paralysis, and so on.

EPIDEMIOLOGY

The precise studies of poliomyelitis in Sweden during the past 70 years have made that country an epidemiological laboratory. The first record was of a small localized epidemic of 18 cases in 1881. Before that the disease had never been recognized as an epidemic possibility.

Table 4, from the monograph of Kling, Pettersson, and Wernstedt (1912), shows the trend of events in Sweden leading up to the epidemic of 1911.

This Swedish pattern of epidemics has been followed in most countries, but it is difficult to give statistical comparisons because even in epidemic years many towns and districts may escape infection, with a flattening out of the incidence when applied to the

whole country. Thus the highest attack rates are seen when a localized community is infected in circumstances favourable to its spread.

TABLE 4

Epidemiological pattern of poliomyelitis, Sweden, 1881-1911

Year	No. of cases	Place
1881	18	Umea
1887	43	Stockholm
1888	14	Ammeberg
1895	21	Stockholm
1899	54	"
1903	20	Gothenberg
1905	1,016	Sweden
1906	429	"
1907	467	"
1908	317	"
1909	178	"
1910	180	"
1911	3,840	"

(Before 1905 total cases for Sweden were not known.)

High attack rates vary from the 240 per 100,000 of population in the Mauritius outbreak of 1945 (McFarlan, 1946), and the 126 per 100,000 in Denmark of 1934 (Jensen, 1934-5), but generally speaking the all-over incidence in big towns does not reach 30 per 100,000 in any epidemic season. In the epidemic year of 1947 the incidence in England was 18 per 100,000. The highest rate previously recorded was in 1938 with its 4 per 100,000. Were it to reach the level of 100 per 100,000 of the population, England would be facing the calamity of 45,000 cases of poliomyelitis in one epidemic season. The epidemic constitution of poliomyelitis in England does not suggest, however, that we shall reach such an extremity in our time.

It is not known what determines the occurrence of epidemics of poliomyelitis. They are irregular in such a way as to suggest the chance that allows the entry into a community of a strain of virus to which that community is susceptible. That strain may be a locally arising mutation as Burnet suggests, or a new strain imported from outside. Given a sufficient number of people susceptible to an invading strain a spread to epidemic proportions may arise.

The rapidity and method of spread show how casual the contact leading to infection may be. The direction of spread along lines of travel was early pointed out in the Scandinavian studies, but whether it spreads by droplet contact or by physical touch is not yet known.

Epidemiological studies in families, schools, and group communities show that in epidemic times most of those who are exposed and non-immune become infected, harbour, and excrete the virus for at least 2 or 3 weeks, but that in the majority no clinical illness ensues. Some may develop subclinical illnesses, others abortive illnesses recognizable as non-paralytic poliomyelitis, and only a few are stricken with the paralysis of poliomyelitis.

However incomplete the epidemiological observations may have been before 1880 in Sweden, or before 1900 in other countries, there is now sufficient evidence to demonstrate that: (1) during the past 70 years there has been an increasing tendency for poliomyelitis to assume epidemic form, (2) the epidemics are limited in season, (3) they are tending more and more to affect the older age-groups, (4) their changing epidemic constitution is operating in the socially advanced countries, (5) England has been remarkably free from big epidemics before that of 1947, and (6) the spread of the infection in epidemic times is more likely to be through the unsuspected healthy carriers in the community than from the patient conspicuously ill with the disease.

INCUBATION PERIOD

We do not know enough to state precisely the incubation period of poliomyelitis. We know that the illness with neck stiffness or paralysis follows 5-10 days after exposure to infection, but the interval may be only 3-4 days or as long as 20-30 days. Aycock (1941), an authority on the subject, gives 6-20 days as the limits. There is something more in this than the process of a statutory incubation period, because the interval between infection and paralysis is so variable. The reasonable explanation is that during the 20-30 days after infection, while the virus is struggling to multiply in the tissues of the body, some secondary factor such as exhaustion, tissue injury, or intercurrent illness predisposes the anterior horn and other susceptible cells to a degree of injury which reflects itself in the clinical illness of poliomyelitis.

THE ILLNESSES OF POLIOMYELITIS

In the period when clinical research was concerned with the detection of physical signs of morbid anatomical lesions it was customary to describe the illnesses of poliomyelitis in topographical terms. There was the common spinal form with flaccid paralysis, the bulbar form, the cerebellar form, and so on. These descriptions served their purpose at that stage of medicine, but gave no picture of the dynamic course of the illness with all its variations. Now that we are more concerned with its early diagnosis and are attempting to understand the time sequence of clinical events, observation and description are directed more to the various methods of onset, the detection of the illness without localized signs, and the succession of events that may lead to paralysis or death. The paramount difficulty in this method of approach is that the main evidence in the pre-paralytic stage of the illness is in the subjective sensations described by the patients. This restricts our understanding and diagnosis of the early stages of the disease in young children unable yet to rationalize their sensations.

It is worth noting, however, that an observant mother often senses that there is something particularly amiss with her young child in the pre-paralytic stage of poliomyelitis. It is a kind of illness she has not seen before. The character of the listlessness, or the type of headache, or the nature of the muscle pain lead her to suspect the unusual. A woman who is not prone to trouble her doctor unnecessarily over her children will often summon him within a few hours of the onset of the child's illness of poliomyelitis. She may be temporarily assuaged by his diagnosis of 'feverish cold' or 'summer influenza', but the doubt and anxiety quickly return unless the initial illness subsides immediately. Or she continues in doubt day by day until the correct diagnosis declares itself with neck stiffness or paralysis.

There are so many variations in the time sequence of the symptoms and the signs of poliomyelitis that it would be true to say that no two patients are ill in exactly the same way. Nevertheless the disease must be described as taking its course within broad recognizable lines, especially in the older child and adult able to describe their symptoms. In the younger child we shall be less sure of our ground, but therein lies the difficulty and fascination of clinical paediatrics.

The Nature of Disease

The common form of the illness of poliomyelitis is best studied and recognized as occurring in four stages, each of which may be an isolated episode or follow in sequence.

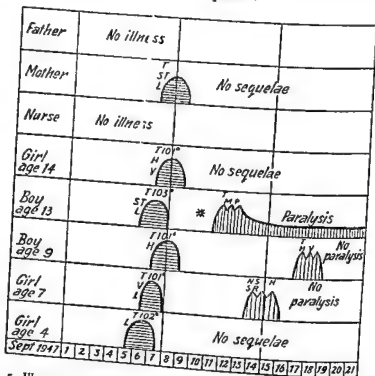


FIG. 5. Illnesses in a family of which one child had paralytic poliomyelitis. The family lived in a remote country house. The possible source of infection was a visitor between 16 August 1947 and 28 August 1947.

T = temperature ST = sore throat L = lassitude H = headaches V = vomiting M = muscle pain NS = neck stiffness SR = spinal rigidity
* = severe muscular exercise by rowing a boat against the wind.

Cross hatching = illness of initial infection.

Vertical hatching = paralytic or other subsequent illness.

1. *The initial illness of infection.* This can be construed as the non-specific febrile illness arising when the virus has multiplied sufficiently in the tissues to produce systemic symptoms. It may not be clinically evident or it may cause an influenza-like syndrome. That may be all there is to it, or it may pass immediately or after an interval of a few days into the other stages.

2. *The stage of muscular discomforts.* Muscle-aching and muscle-stiffness are the chief symptoms. The disease may abort at this stage or progress to paralysis.

3. *The neck-stiffness stage.* This so-called meningitic stage may be indistinguishable from other forms of aseptic meningitis, but would be strongly suspicious of poliomyelitis if it occurred in epidemic seasons.

4. *The paralytic stage, or the stage of visible clinical effect in the central nervous system.* In this schematic description the term 'abortive poliomyelitis' is avoided. It is a confusing term, because the disease may abort at each stage in the sense that recovery may then take place without permanent paralysis or death. When there is an interval of apparent recovery between stage 1 and stage 3 or 4, the illness is described as of the 'dromedary type' because of the biphasic rises of temperature. The first three stages, if they can be diagnosed, may be regarded as pre-paralytic poliomyelitis. These stages do not follow in sequence in all patients. Occasionally paralysis may appear in one group of muscles, to be followed by stiffness in another.

The initial illness of infection

With some suddenness, as, for example, in the course of a morning, the patient previously in good health feels tired and languid. There may be headache within a few hours, but this is not at first remarkable, and in the absence of any localized discomfort the patient tends to proceed with his play or occupation. Some young adults, not having felt such symptoms before and not feeling unduly ill, go for a walk or take other exercise to 'work it off'. The temperature rises, but they sleep fairly well that night. If there are localized symptoms at this stage they take the form of a peculiar feeling in the throat which patients describe as 'a sore throat but not sore like tonsillitis', or slight headache, or they may vomit. Diarrhoea, described in many textbooks as an early symptom, may occur, but is infrequent in my experience.

Already towards the end of the first day muscular discomforts may arise. These vary from a feeling of muscle-aching, such as might be felt after muscular exercise, to severe muscular pains, often described as spasms or cramps. The patient's attitude at this stage is usually one of contented apathy combined with mental clarity.

If the initial illness is an isolated episode not leading to the later stages of the disease it terminates within 36-48 hours. Its epidemiology in relation to paralytic poliomyelitis has been studied by Ryle (1948), Casey, Fishbein, and Abrams (1947), and others. If the true nature of the initial illness is not recognized it may be misdiagnosed as 'summer influenza'.

The stage of muscular discomforts

This may follow immediately on the initial febrile illness, or appear after an interval of apparent recovery, or it may be the first evidence of the disease. The muscle discomforts of poliomyelitis are something more than the vague general achings of a non-specific febrile illness. They may be localized in groups of muscles or affect so many groups as to appear widespread. They may appear as backache, or pains in the chest, or shoulder pain, or pain in the neck, or aching in the thighs, and so on. There is no evidence that if paralysis follows it will affect the most painful muscles. In severe forms the discomforts increase to pains, which fluctuate in intensity, and unless there are other diagnostic features I find them indistinguishable from the pains of Bornholm disease (epidemic myalgia).

With the muscle pains there may be alteration in tone to resemble spasm. Caughey and Malcolm (1950) suggest that this is a constant feature of poliomyelitis, having found it in all the cases they saw in the New Zealand epidemic. Although it may appear to be mere argument about words, spasm hardly seems to be a true description of the condition. The muscles are stiff and they resist passive stretching. If the spinal muscles are affected the patient cannot bend forward and assumes the tripod position on sitting up. If the muscles of the shoulders are affected there is updrawing and rotation forward of the shoulders.

These observations on muscle stiffness in poliomyelitis have amplified the clinical picture of the disease. It is no longer sufficient to examine only for neck stiffness or hamstring rigidity. In each case a systematic examination of all groups of muscles should be carried out, and cases will be found who have isolated muscle stiffness in sites such as the feet or biceps, but without neck stiffness.

This stage of muscle pain and stiffness may last for a day only or for up to 2 weeks. I have known a child with stiffness so marked as

to resemble a decerebrate rigidity, and lie with it for 2 weeks and then make a recovery without paralysis.

In this stage the cerebrospinal fluid usually shows the changes of poliomyelitis with its slight pleocytosis and increased albumin content. At the same time early localizing neurological signs from lesions in the cord or brain-stem may appear, with a slight facial weakness, a unilateral weakness of the palate, or a peculiar unsteadiness of the eye which looks like nystagmus but has been better termed 'refixation tremor' or ocular 'myoclonic ataxia'. Similar to this is a peculiar unsteadiness in gait which may precede the actual paralysis or weakness of the legs.

The neck-stiffness stage (meningitic poliomyelitis)

Neck stiffness is a common feature of poliomyelitis. It may occur in patients who may thereafter recover without paralysis, and with it there is usually also spinal rigidity. It may be so slight in degree as to require careful search. It may be immediately evident so as to suggest meningitis. It may be so extreme as to cause alarming opisthotonos. In a young child with poliomyelitis seen in the summer of 1950, the stiffness and retraction arched the spine and neck until there was a space of only $5\frac{1}{2}$ inches between the occiput and the sacrum.

Even without lumbar puncture the differential diagnosis from meningococcal and other forms of meningitis will be suggested because in poliomyelitis the patient's mind is usually clear. He may lie in an agony of discomfort from the head retraction yet remain capable of discussing his symptoms alertly and precisely.

Examination of the cerebrospinal fluid usually shows the characteristic pleocytosis and increase of protein, but these changes do not run parallel to the degrees of neck stiffness. With extreme neck retraction there may be no more than 50 cells per cubic millimetre. With a minimal degree of neck rigidity there may be 500 cells or more. Nor do the changes in cerebrospinal fluid run parallel to the duration of the rigidity. A patient may have extreme rigidity and retraction of neck and spine for more than a week with minimal changes in the fluid.

This raises again the question of nomenclature. Should this form of poliomyelitis be called meningitic? Is the neck stiffness and retraction a result of inflammation of the meninges? There is reason to doubt this. If the signs were due to meningitis only, we

would expect the degree of stiffness to be in proportion to the pleocytosis. It would be better to regard the picture as we regard the stiffness and retraction of other muscles. While they are all part of the same process, the stiffness is more consistently present and more easily seen in neck and spinal muscles than elsewhere.

The search for neck stiffness is one of the classical methods of examination in a suspected case of poliomyelitis. When it is slight it will require the practised hand to demonstrate it. The examination should be reinforced by asking the patient to sit up and touch his knees with his nose. If there is spinal rigidity he will be unable to do this, and will assume the tripod position in which his trunk fails to reach the vertical and he props himself up with his arms behind him. The examination should include also a search for similar changes in other muscles of limbs, trunk, and neck for signs of paralysis, and for cranial nerve disorders including the ocular signs already mentioned.

Paralytic poliomyelitis and other clinical effects in the central nervous system

In the spinal cord. The classical feature of poliomyelitis is the lower motor neurone paralysis resulting from damage or death of the anterior horn cells of the spinal cord. The paralysis affects groups of muscles, and many groups of muscles may be affected or single muscles within a group. Very occasionally slight paraesthesia of the skin may precede or accompany the paralysis, but in general it is a motor paralysis of the flaccid type without sensory changes.

The legs are more frequently attacked than the arms, the arms more frequently than the trunk, the trunk and intercostals more frequently than the neck. In the legs the quadriceps and peronei, and in the arms the deltoid are more frequently paralysed than the other muscles. But extraordinary combinations of muscle-group paralysis occur. I have seen a boy with all the muscles of one arm permanently paralysed, while the rest of the body escaped.

A hundred years ago West called the disease 'morning paralysis' because, so he said, the paralysis would be discovered unexpectedly on waking without previous warning. That was, surely, a faulty observation. There is in most cases some preceding illness or symptom, however slight or transient, before paralysis appears.

If the patient has been in bed for a few days with an initial illness the paralysis may not be evident until he gets up. It is therefore a

good rule when visiting a child with an unexplained febrile illness to turn back the bed-clothes and ask him to dorsiflex his feet, to raise his legs, to sit up, and to raise his arms to see whether there is paresis anywhere. He should be asked also to cough to see whether there is weakness of the intercostal muscles or diaphragm.

The time sequence of paralysis is a very important aspect of the disease. It may appear within 24 hours of the onset of the illness. It may be delayed for more than 10 days. Generally speaking it is evident on the third or fourth day of the illness. If there is a progression of paralysis from one group of muscles to another, or from the legs to the trunk, from the trunk to the arms, and from the arms to the bulbar muscles, it is usually a quick sequence of events occurring within 2 or 3 days. So the extent of the paralysis can be foretold at the end of that time in most cases. But this is not an absolute rule, and cautious expectancy should be exercised for a week before pronouncing on the finality of the paralysis. The degree of paralysis varies from a slight paresis with the loss of deep reflexes lasting a few weeks to the most complete flaccid paralysis which is permanent. Although the paralysis is usually asymmetrical, it occurs sometimes as a complete paralysis of both legs resembling a transverse myelitis.

Disturbances of micturition are rare during the paralytic stage, but Lawson and Gravey (1947) claim that, if looked for, transient bladder paralysis in the early stages of the illness will be found in 10-30 per cent. of cases.

The relation of the site of paralysis to previous muscle injury, exhaustion, tonsillectomy, muscle injections, pregnancy, and other aetiological influences will be discussed later.

Bulbar paralysis in poliomyelitis. Most of the deaths from poliomyelitis result from bulbar paralysis. That alone raises this form of the disease to a high level of clinical importance. But the patients with slight degrees of bulbar paralysis, from which they recover, so often evince the anxiety of death that it must always be regarded as a medical emergency.

Wickmann's classical estimate of a 6 per cent. incidence of bulbar paralysis in poliomyelitis understates its frequency if we include all cases of slight unilateral weakness of the palate, but it is near the truth for the severe forms.

The symptoms of bulbar paralysis vary in time sequence and severity. They vary also according to the anatomical localization

of the nerve-cell damage. The most severe form is that resulting from paralysis of the lower cranial nerves concerned in swallowing. If this is accompanied by weakness of intercostals and diaphragm there is increased danger of inhalation pneumonia. Difficulty in swallowing saliva is the outstanding symptom. With it there may be laryngeal paralysis or weakness of the tongue. The threat to life depends mainly on the degree to which the tenth nerves are involved. Unless the pooling of the saliva in the pharynx can be released the patient will die of suffocation. This raises the question of tracheotomy in these patients, concerning which method of treatment there are two schools of thought. So dire a step should not be necessary if other measures are used to drain the saliva and if tube feeding is used.

It will be appreciated how important it is to distinguish the respiratory distress due to the suffocation of bulbar paralysis from the paralysis of the respiratory muscles. Otherwise an artificial respirator may be used in the wrong type of case and hasten death.

Death from bulbar paralysis is not always due to the mechanical suffocation from tenth-nerve paralysis. With relatively slight paralysis of the swallowing muscles, distress or death may take place in such a manner as to suggest involvement of the vital centres of respiration and circulation in the medulla. To detect this again needs accuracy and discrimination in order to avoid doing harm by faulty treatment.

Paralysis of the upper cranial nerves is less alarming than lower bulbar paralysis. The group includes the motor division of the fifth nerve, the sixth, seventh, and eighth nerves, as well as the ocular nerves. The encouraging feature about poliomyelitis of the upper cranial nerves is that the resulting paralyses are rarely permanent.

In the eyes there may be ptosis, paralytic squint, or other oculomotor weakness. The symptom of 'refixation tremor' of the eye (Strickland, 1947) appears to be the same as the 'opsoclonia' of Marmion and Sandilands (1947). This is a jerky movement of the eyes before they come to rest when focusing on an object directly in front or slightly to the side.

Although there is a great variety of symptoms and their severity in bulbar poliomyelitis, the most dramatic cases are those with rapid development of paralysis early in the illness, whether it arises as the primary neurological symptom or follows paralysis of limbs and trunk. These patients may die within 3 or 4 days, and it is a

terrifying mode of death because the patients may remain conscious until the end. They watch themselves suffocate.

The incidence of bulbar poliomyelitis rises with the age of the patients. For that reason the disease is more likely to kill the adult than a young child. The trend of study into the links between paralysis and the precipitating aetiological factors has not explained this correlation of age with bulbar paralysis, but it is tempting to suggest that adults talk and swallow too much.

Cerebral and cerebellar poliomyelitis. If a patient ill with poliomyelitis becomes drowsy or comatose it is often concluded or guessed that he has cerebral poliomyelitis, especially if he has neck stiffness, cranial-nerve palsies, and the expected changes in the cerebrospinal fluid. Here, however, two immediate distinctions should be made. Cerebral symptoms may arise in poliomyelitis from cerebral anoxia, if there is respiratory paralysis. The symptoms are then due to the anoxia and not to an extension of the disease to the cerebral hemispheres. Secondly, even in epidemics of poliomyelitis, other viruses may be prevalent which will cause encephalitis. For example, the virus of mumps may cause encephalitis without swelling of the parotid glands or other clinical evidence. Nevertheless cerebral poliomyelitis does occur with a variety of symptoms dependent on the portions of the cerebral hemispheres affected. Such cases have been confirmed by virus studies (Kelleher, Bratton, and MacCallum, 1949).

Apart from cerebral poliomyelitis with its classical signs, other minor and transient cerebral symptoms may arise. I have known a meek and serene child develop violent temper outbursts and other behaviour faults during the first few days of the illness. Vouched for by an observant mother they could have no other explanation than organic cerebral damage. More often the mental and emotional state is one of contented apathy, unless the muscle stiffness is painful. To describe this feeling, young adults, who are able to analyse their sensations, often comment on the peculiar character of this contented apathy. In it they are disinclined to argue or to fuss while remaining alert and mentally clear. These feelings become disturbed only as the fear of paralysis strikes them. It is possible that these transient symptoms denote a hypothalamic disorder.

Poliomyelitis with a clinical picture of excessive drowsiness or coma resulting from the cerebral lesions is comparatively rare. Of

184 patients with poliomyelitis in the epidemic of 1947 I saw only one patient in this condition and the diagnosis was not confirmed by virus studies. Hemiplegia has been described as a result of poliomyelitis, but I know of no case confirmed by laboratory tests.

Recognizable cerebellar symptoms of polioencephalitis are commoner than cerebral symptoms. A child's paralytic illness may be preceded by alterations in gait suggesting this possibility. Very rarely does it reach a stage of gross cerebellar ataxia. More often it shows itself by a peculiar dithery gait, a jerky unsteadiness in walking, lasting for a day or so before the paralysis strikes and obscures it. The arms may be similarly affected with an unsteadiness as a grasp is attempted. As in cerebral poliomyelitis there is usually other clinical evidence to suggest the diagnosis, but full confirmation depends on epidemiological or laboratory evidence.

The course of the illness of poliomyelitis

The classical illness of poliomyelitis is that of a young child overcome with fever and lassitude but without cough, coryza, rash, or other localizing symptoms. The diagnosis is in doubt and remains in doubt for a day or two. Then, either immediately or after a period of recovery lasting up to 3 or 4 days, the illness relapses with severer symptoms. The temperature rises again. The child lies quietly. Headache appears, especially with the jar of walking down stairs. On examination at this stage, that is on the second or third day of the major illness, paralysis in a group of muscles in arm or leg is noted. The diagnosis is now evident, and the fever subsides. Closer examination now reveals a little neck rigidity, and the cerebrospinal fluid is found to be clear, but to contain 50-200 cells and an increased amount of protein. The paralysis does not extend beyond that seen on its third or fourth day, and thereafter begins to improve within a few weeks or remains permanent in spite of all therapy.

There are many variations of this standard course of the illness of poliomyelitis, and the age of the patient plays some part in determining them. In a young infant the initial illness may be so slight as to be easily overlooked, and paralysis in an arm or leg is the first evidence of the disease. Or the illness may merge into violent neck retraction or stiffness of hamstrings or other muscles.

The illness of poliomyelitis which does not result in paralysis, often described as 'abortive' or 'non-paralytic' poliomyelitis, may

vary from a symptomless feverish illness lasting 24-48 hours to an illness lasting 2 weeks with disabling neck retraction and muscle stiffness.

The progress of paralysis varies also. It may go no farther than the paralysis of the first day or it may spread to other muscles in other limbs or in the trunk over a period of 6 or 7 days. If the intercostals are affected weakness of cough will reveal it. The clue to the ultimate course of the illness, in terms of permanent paralysis, can be judged from the extent, the site, and the completeness of the paralysis at the end of the first week's illness. The most crippling of the permanent paralyses are those affecting the trunk and thigh muscles.

PROGNOSIS

Accurate statistical estimates of death in disease are interesting, but may be unhelpful in clinical practice. If a disease has an estimated mortality of 10 per cent. the patient under our care is not solaced by these estimates. He wishes to know if he is in the 10 per cent. or the 90 per cent. So prognosis in the practice of medicine is a matter of experience of many individual illnesses. Moreover, statistical accuracy is difficult because of the great discrepancies in diagnosis in different series of cases.

The broad features of prognosis in poliomyelitis are indicated from a sample from the epidemic in Great Britain in 1947. In the period July to December the age distribution of deaths registered were as follows:

<i>Age in years</i>	<i>Deaths</i>
0-4	127
5-14	149
15-24	151
25-34	138
35-44	60
45 onwards	24

In this epidemic 58 per cent. of the deaths registered were of persons over 15 years, whereas only 33 per cent. of the notifications were in that age-group.

In New York in 4,215 cases of the epidemic of 1916 the fatality rate was 21.4 per cent. In 1930 in 660 cases it was 16.8 per cent. In

1931 with 2,051 cases it was only 8 per cent. Even these figures convey little information about prognosis unless we know that statistics from different epidemics are comparable by including non-paralytic cases on a standardized basis of diagnosis.

Second attacks of poliomyelitis

In 1930 Still recorded the case of a girl with a second attack of poliomyelitis with an interval of 5 years between the attacks. He collected 8 other recorded cases. This phenomenon can be explained on the assumption that infection from one strain of virus may fail to immunize against another strain.

TREATMENT

There is neither a preventive nor a curative treatment for poliomyelitis which is specific for the disease. The best that can be done is to diminish the chance of infection in epidemic times, to reduce the risk of paralysis if infection takes place, and to control the effect of paralysis if it occurs. In the case of permanent paralysis, technical orthopaedic treatment and advice will be required to aid the crippled patient.

While the technical aspects of preventive and curative treatment may absorb the interest of doctors and nurses, it is of paramount importance that they should apply their skill in such a way as does not diminish the morale and fortitude of the patient. A boy may be paralysed in the legs by poliomyelitis and for this doctors and nurses may be able to do little. But in attempting what they can do they should not paralyse his spirit. The handling of a young person who is paralysed or threatened with paralysis requires that insight and common sense which comes within the scope of good doctoring and good nursing.

Preventive treatment

There is as yet no vaccine or other specific preventive treatment for poliomyelitis. On theoretical grounds, and in the light of our present knowledge, the only feasible step would be to expose young infants to infection in early infancy and run the risk of paralysis for the sake of immunization, but this is not a practical measure.

In epidemic seasons prevention should aim at avoiding spread of infection from one community to another. It were better at such

times that children should stay at home idling about, than endure the risks and exhaustion of travel.

Within a community where a case of poliomyelitis has occurred children should avoid abnormal exertions, and chances of faecal cross contamination should be controlled. Swimming, which combines excessive muscular exercise and exposure to cold, should be banned except to those who are regular in this habit. School sports and other exercises involving new muscular activities should be postponed. It is difficult to strike a happy medium between excessive valetudinarism and cavalier carelessness, but a lesson can be learned from the case of the medical student who sits his examinations after many sedentary weeks, and then goes to a violent rowing match without previous training and in spite of having felt ill and feverish the day before, and in spite of the poliomyelitis prevalent in his village, and then dies of poliomyelitis within 5 days.

The best that can be done in the pre-paralytic stage is to keep the patient in bed and make him warm and comfortable. If there is painful muscle spasm local heat should be applied. This was the basis of the treatment advocated by Sister Kenny. It was thought that early application of hot packs would diminish the spasm and thereby decrease the subsequent paralysis. While these local applications may comfort the patient there is no evidence that they prevent the paralysis. Nevertheless it is treatment in the right direction and Sister Kenny deserves great credit for her attempt to lift the early treatment of poliomyelitis beyond the mere use of mechanical manipulations. Her concept also of 'mental alienation' has caused the medical profession to think again about the management of patients with paralysis.

Treatment of paralysis

If muscles paralysed by poliomyelitis are going to recover they will begin to do so within 2 months, and most of the worthwhile recovery will take place within 4 months (Harry, 1938). As soon as the early illness of poliomyelitis is over, and the extent of the paralysis is known, the cure of the patient and the treatment of the paralysis can be planned.

Seddon (1947, 1948) has given practical advice on the aims and techniques of treatment. The first aim is the prevention of faulty posture and overstretching of affected muscles. The legs can usually

be maintained in a correct position by means of small pads under the knees and by supporting the feet at right angles with boards. Cradles and splints may be required in special circumstances, but their use must be guided by reason and experience.

Preventive treatment in the pre-paralytic stage

If, in the summer season, a person has an undiagnosed febrile illness or symptoms which in any way suggest the initial illness of poliomyelitis, he should go to bed and stay there until he feels quite well. If the illness is accompanied by muscle stiffness or by other signs highly suspicious of poliomyelitis, the patient should be confined to bed until all danger of paralysis is past. If this is impossible he should at least limit his activities and avoid any exhaustion.

There was a time when treatment with convalescent poliomyelitis serum was tried in the early stage of the illness, but there is no evidence that it had any effect. Dehydration therapy and cerebrospinal fluid drainage have also been advocated on the hypothesis that the oedema of the brain and cord would be reduced by this treatment. There is no evidence that it did good. It may have done harm.

Passive movements of the paralysed parts will help to prevent contractions. They can be used twice a day as soon as the acute stage of the illness is over, even if a little painful spasm still persists. If early passive movements increase or cause pain, they should be gently applied and helped by the preliminary application of hot packs. Massage is a ritual long in use for the treatment of paralysed muscles. It is difficult to see its value beyond that of encouraging the patient.

While most of the recovery from paralysis takes place spontaneously, the severe cases will be helped by re-education of muscles and by under-water exercises. The latter are especially valuable in the treatment of paralysis of the trunk and leg muscles. In re-education the physiotherapists will have three aims: (1) re-education of individual muscles, (2) the practice of complex movements, and (3) the development of trick movements when it is apparent that recovery is reaching a standstill. As an example of a trick movement Seddon cites the use of the common flexor and extensor muscle masses as effective flexors of the elbow when the biceps and brachialis are completely paralysed.

In some hospitals and clinics it has been customary to keep patients with leg and spinal muscle paralysis in bed for 12-18 months. This has been done in the belief that such small returns in power as may occur are worth waiting for. There is now a reaction against this excessive caution. Patients are encouraged early to attempt to walk because the attempted resumption of normal posture is likely to be more valuable than the little improvements that may occur during the later months of prolonged recumbency. On this Seddon (1947) writes: 'at this stage it is impossible to say which practice is right, the old one or the new, but it seems likely that in the past we have erred on the side of excessive caution, perhaps to the detriment of limbs which, if more activity had been allowed, would have suffered less from the effects of general atrophy.'

EXPLANATION AND ADVICE TO PATIENTS

Late one night I was summoned to a consultation on a young woman ill with pneumonia. Recognizing me as I entered her room she turned eyes of hatred and venom on me, much to my discomfort. Six years before she had been ill with poliomyelitis and in that illness I attended her. She had survived to become a hopeless cripple with paralysis of trunk muscles and great deformity of the thorax. From one institution to another she finally passed to the care of her mother, a woman with too little insight and too little help from her neighbours to enable her to manage the situation. The girl's life was now all misery and recrimination, and she complained that she had been allowed to live. She blamed me on the one hand because I had failed to explain and had given her false hope, and on the other because I had saved her life. The first of these may have been true. The second certainly was not. I had done little more than diagnose the disease. She hated me when I came back six years later, because she feared that I would prolong her life a second time, when she wished only to die.

This example is unusual. The wretched young woman was an unhappy and perhaps abnormal person. But it serves to remind us that a great deal of the practice of medicine consists in giving the right kind of explanation and advice to people who are ill or imagine themselves to be ill. What should be told to a patient paralysed with poliomyelitis? When shall it be told? How shall it

be told? So much publicity and hysteria hangs on the spectre of poliomyelitis that this responsibility of explanation must be carefully considered. Explanation is particularly desired by the older child, the adolescent, and the young adult who is sensitive and intelligent. If they are ambitious also, or intolerant, or cruel, they will require careful handling in order to mitigate the effects of the new experience on their personalities. There is no cruelty like the cruelty of a discontented cripple, as there is no serenity of mind like that of a man or woman who rises above his or her deformity. *But they need help to rise.*

While no general rules can be laid down for so individual a responsibility it is in most cases wise not to delay full explanation of the paralysis beyond the stage when it seems to be permanent. Most human beings, if told in the right way and at the right time what is in store for them, will rise to the occasion. So in his illness a patient paralysed by poliomyelitis requires more than his technical treatment and occupational therapy. He should be told not too late what degree of physical bankruptcy he must expect, while at the same time he is advised how he will arrange his life in spite of it. But he must be told by someone educated and skilled in the art of explanation and advice.

REFERENCES

- AYCOCK, W. L. 1941. *New Engl. J. Med.* 325, 1941, p. 405.
 BASKIN, J. L., and others. 1950. *Amer. J. Dis. Child.* 80, 1950, p. 10.
 BIERMANN, A. H., and PISZCZEK, E. A. 1944. *J. Amer. med. Ass.* 124, 1944, p. 296.
 BODIAN, D., and HOWE, H. A. 1945. *J. exp. Med.* 81, 1945, p. 255.
 BURNET, F. M. 1945. *Virus as Organism*, Cambridge, Mass., Harvard University Press, 1945.
 ——— 1948. *The Background of Infectious Diseases in Man*, Melbourne, 1948.
 ——— and others. 1939. *Aust. J. exp. Biol. med. Sci.* 17, 1939, p. 375.
 CASEY, A. E., and others. 1947. *Amer. J. Dis. Child.* 72, 1947, p. 661.
 CAUGHEY, J. E., and MALCOLM, D. S. 1950. *Arch. Dis. Childh.* 25, 1950, p. 15.
 DALLDORF, G., and SICKLES, G. M. 1948. *Science*, 108, 1948, p. 61.
 ——— ——— 1949. *J. exp. Med.* 89, 1949, p. 567.
 FABER, H. K., and SILVERBERG, R. J. 1946. *Ibid.* 5, 1946, pp. 78, 499.
 FLEGNER, S., and LEWIS, P. A. 1909. *J. Amer. med. Ass.* 53, 1909, pp. 1639, 1913, 2095.

- FRANCIS, T., and others. 1942. *Ibid.* 119, 1942, p. 1392.
- GARD, S. 1938. *Bull. Off. int. Hyg. Publ.* 29, 1938, p. 269.
- GEFFEN, D. H. 1950. *Med. Offr.* 83, 1950, p. 137.
- HARRY, N. M. 1938. *Brit. med. J.* 1, 1938, p. 164.
- HORSTMANN, D. M. 1948. *Lancet*, 254, 1948, p. 273.
- and others. 1944. *J. Amer. med. Ass.* 135, 1944, p. 11.
- HOWE, H. A., and BODIAN, D. 1942. *Neural Mechanisms in Poliomyelitis*, New York, New York Commonwealth Fund, 1942.
- and others. 1945. *Bull. Johns Hopk. Hosp.* 76, 1945, p. 19.
- INTERNATIONAL COMMITTEE. 1932. *Poliomyelitis*, Baltimore, Wilkins & Wilkins, 1932.
- JENSEN, C. 1934-5. *Proc. R. Soc. Med.* 28, 1934-5, p. 13.
- KELLEHER, W. H., and others. 1949. *Brit. med. J.* 2, 1949, p. 213.
- KLING, C., and others. 1912. *Investigations in Epidemic Infantile Paralysis*, Report to 15th International Congress of Hygiene, Washington, 1912.
- LANDSTEINER, K., and POPPER, E. 1908. *Wien. Klin. Wschr.* 21, 1908, p. 1830.
- LAWSON, R. B., and GRAVEY, F. K. 1947. *J. Amer. med. Ass.* 135, 1947, p. 93.
- LEVINSON, S. O., and others. 1945. *Amer. J. Hyg.* 42, 1945, p. 204.
- MCCLOSKEY, B. P. 1950. *Lancet*, 258, 1950, p. 659.
- McFARLAN, A. M. 1946. *Proc. R. Soc. Med.* 39, 1946, p. 323.
- MARMION, D. E., and SANDILANDS, J. 1947. *Lancet*, 253, 1947, p. 508.
- MARTIN, J. K. 1950. *Arch. Dis. Childh.* 25, 1950, p. 1.
- MAXWELL, D. M. W., and WILLCOX, P. H. 1947. *Lancet*, 253, 1947, p. 353.
- MEDIN, O. 1890. *Hygiea, Wien.* 52, 1890, p. 657.
- PAUL, J. R., and others. 1939. *Science*, 90, 1939, p. 258.
- RHODES, A. J. 1947. *Bull. Hyg. Lond.* 22, 1947, p. 353.
- ROMER, P. H. 1909. *Münch. Med. Wschr.* 56, 1909, p. 2505.
- 1910. *Ibid.* 57, 1910, p. 229.
- 1913. *Epidemic Infantile Paralysis*, London, John Bale, Sons & Danielsson, 1913.
- RUSSELL, W. R. 1949. *Brit. med. J.* 1, 1949, p. 465.
- RYLE, J. C. 1948. *Lancet*, 254, 1948, p. 945.
- SABIN, A. B. 1941. *J. Amer. med. Ass.* 117, 1941, p. 267.
- SEDDON, H. J. 1947. *Brit. med. J.* 2, 1947, p. 319.
- 1948. *Practitioner*, 160, 1948, p. 175.
- STILL, G. F. 1930. *Arch. Dis. Childh.* 5, 1930, p. 295.
- STRICKLAND, B. 1947. *Lancet*, 253, 1947, p. 369.
- TIEHLER, M. 1934. *Science*, 80, 1934, p. 122.
- WICKMANN, J. 1907. *Beiträge zur Kenntnis der Heine-Medinischen Krankheit*, Berlin, Karger, 1907.
- WENNER, H. A. 1946. *Yale J. Biol. Med.* 18, 1946, p. 281.

PART II

The Study of Disease

VIII. *Some Observations on Sugar Tolerance, with Special Reference to Variations found at Different Ages*¹

SINCE the advent of practical methods of micro-chemical analysis, blood-sugar estimations have been established as a clinical procedure. As a result of this, much attention has been directed during the past decade to the study of the variations of the glucose content of the blood both in health and in disease. The normal level is known, and many of its physiological variations are recognized. The most important result has been a recognition of the fact that the blood-sugar of a healthy individual reacts in a definite manner to the ingestion of carbohydrate. It is known that this reaction, to which the term 'alimentary hyperglycaemia' (Bailey) has been applied, becomes abnormal in the presence of certain conditions and diseases which affect sugar tolerance or interfere with carbohydrate metabolism. The changes in blood-sugar content consequent on the ingestion of carbohydrate may be expressed as a blood-sugar curve.

In the further investigation of cases of glycosuria, in the early diagnosis of diabetes mellitus and in its differentiation from renal diabetes, blood-sugar curve determinations are of undoubted value. But an extension of the test to other diseases has yielded inconsistent and contradictory results. Certain investigators have claimed that characteristic alterations of the normal blood-sugar curve occur in nephritis, cancer, affections of the ductless glands, and many other conditions. Others have claimed that in no disease is there a specific type of curve, and that its use as a diagnostic measure is of little value. Before these claims can be finally decided, it appears desirable that further investigations should be made into the factors which influence the curve in the normal person. It was for this purpose that the work described in this paper was undertaken. Experiments were carried out to determine whether the response to the blood-sugar curve test was the same at all ages, by

¹ Published in *Quarterly Journal of Medicine*, 14, no. 56, 1921, p. 314.

comparing the curves of infancy and childhood with those of adult life and old age.

So much independent work has been done in different countries that a brief review of the published results may serve to show how much of the knowledge that has accrued can be accepted and put to a practical use. Baudouin in 1908, and Frank in 1910, estimated the blood-sugar of healthy men before and 1 hour after the ingestion of 100 gm. of sugar, and found it to be raised in some cases and unaltered in others. The greatest rise observed was one of 0.03 per cent. Tachau, in 1911, made further observations on healthy cases, and also on cases of diabetes, chronic nephritis, and jaundice. He estimated the blood-sugar at hourly intervals after the ingestion of 100 gm. of glucose. With such infrequent estimations he failed to detect the rise and fall which usually occurs within the first hour, and concluded that, in the healthy person, there was no rise in the blood-sugar after taking glucose. In each of the cases of diabetes he found hyperglycaemia after 1 hour. In 3 of the 5 cases of chronic nephritis which he investigated, he found no rise in the blood-sugar 1 hour after taking the glucose; in the other 2 there were rises from levels of 0.104 and 0.097 per cent. before taking the glucose, to 0.216 and 0.212 per cent. 1 hour later. The work of Rolly and Oppermann, and of Bing and Jakobsen, gave inconsistent results, for they also did not realize the importance of frequent estimations.

It was Jacobsen, using Bang's method of blood-sugar estimation, who pointed out that the blood-sugar began to rise within 10 minutes of the ingestion of a meal of carbohydrate, and, reaching its maximum level within 30 minutes, the hyperglycaemia passed off in many cases by the end of the first hour. It was now evident that if the course of the blood-sugar curve were to be accurately plotted out and used as a test of sugar tolerance, or as an indication of the functional activity of the carbohydrate storage mechanism, attention must be given to these three points: (a) a dose of sugar sufficient to provoke the maximum rise must be given; (b) estimations of the blood-sugar must be made at frequent intervals so that the highest point reached can be detected; (c) the estimations must be continued until the blood-sugar returns to its normal or fasting level.

Sakaguchi's careful work covered this ground. Using Bang's method, he estimated the blood-sugar at intervals of 10 minutes

for 2 hours after the ingestion of 100 gm. of glucose in healthy young adults. His results are very similar to those of Jacobsen. The rise in blood-sugar was appreciable within 10 minutes. It continued, reaching its maximal height within 20 to 40 minutes, and subsided quickly, returning to or below its fasting level from 50 to 90 minutes after the sugar had been taken. He found that the highest point of the curve varied between 0.133 per cent. and 0.191 per cent., but the rapid subsidence after reaching this point was constant in all his experiments. This requires emphasis, for it is this rapid return of the blood-sugar to its original level that denotes the efficiency of the carbohydrate storage mechanism; while the actual height to which the curve can rise may vary in the healthy organism with normal sugar tolerance as much as 0.050 or 0.060 per cent.

Hopkins, Hamman and Hirschmann, Bailey, and others, by a combination of blood and urine analyses have shown the relation between hyperglycaemia and glycosuria. There is substantial evidence in their work that in the normal adult the kidney threshold level for sugar is reached when the blood-sugar rises to a level of about 0.17 per cent.; and that it is at that level that active secretion of sugar by the kidney takes place.

This knowledge of the level of the renal threshold has been of value in investigating those cases of glycosuria to which Klemperer gave the name 'renal diabetes', and Saloman 'diabetes innocens'. Graham has contributed a paper on this subject in which 5 cases of diabetes innocens are described. Blood-sugar estimations served to differentiate them sharply from diabetes mellitus. There was no hyperglycaemia; the blood-sugar curves were normal or lower than normal, yet sugar was secreted actively by the kidneys. Goto has reported another case. His paper contains a full review of the literature on this subject.

The use of the determination of the blood-sugar curve as a clinical test is described very clearly and fully in a paper by MacLean and de Wesselow. They compared the curves of normal adults with those of diabetics. They determined the response to various carbohydrates, using glucose, cane sugar, laevulose, lactose, galactose, maltose, and potatoes. Each of these provoked a somewhat similar rise except laevulose, the ingestion of which had no effect upon the blood-sugar. It was shown in their experiments that a dose of 25 gm. of sugar was sufficient, in the healthy

organism, to produce a maximal rise, usually from a fasting level of between 0.09 and 0.11 per cent. to a height of 0.17 per cent., and that larger doses were incapable of making the blood-sugar exceed this level, serving only to prolong slightly the descent of the curve. They enunciate the working rule that if the blood-sugar fails to return to its original level within one and a half hours after a single dose of 50 gm. of glucose, a defect in the carbohydrate storage mechanism may be considered to be present. They showed that the characteristic of the diabetic curve is that it continues to rise for 2 or 3 hours after the ingestion of glucose, and is followed by such a slow decline that at the end of 4 or 5 hours it still may be raised above the original level. In cases of severe diabetes the rise in blood-sugar may be to levels of 0.300 or 0.400 per cent., while the milder cases of diabetes would present blood-sugar curves which approximated more to those of a normal person.

This prolonged hyperglycaemia in diabetics following the administration of a dose of glucose has been noted by most of the workers on the subject, and there is general agreement as to the essential difference between the normal blood-sugar curves and those of a diabetic. But as yet no satisfactory explanation has been put forward as to why, in the healthy person, the hyperglycaemia should pass off, and the blood-sugar curve return to its original level, at a time when absorption of the glucose from the alimentary canal is still proceeding. That this is the case, absorption continuing after the normal blood-sugar curve has returned to its original level, is apparently true, for it is revealed in the severe diabetic by the steady rise in blood-sugar which continues for 2 or 3 hours after the ingestion of glucose. The hypothesis of MacLean and de Wesselow is that the drop in the normal curve is caused by the sudden intervention of a storage mechanism, which abstracts the sugar from the blood more quickly than it enters. Hamman attempted to throw light on this point. He found that the administration of a second dose of sugar immediately after the reaction of the blood-sugar to an earlier dose was complete did not cause so great a rise in the blood-sugar level as the first dose. The difference between the first and second reactions was slight, but was constant in all 9 cases investigated. He assumed that the first dose of glucose stimulated the mechanism of carbohydrate disposal so that the repetition of an equal dose while this mechanism was still in action produced a less marked hyperglycaemia.

It has been suggested by Fisher and Wishart that this sudden fall in the blood-sugar level is due to a change in the blood-volume, the osmotic effect of an increased sugar content producing a dilution of the blood. Bailey investigated this point in subsequent work, and found only slight changes in the blood-volume as measured by haemoglobin estimations. Strouse attempted to change the blood-volume by varying the water intake, and by administering cathartics; he found that these measures had no effect on the blood-sugar level. Both of these authors conclude that the sudden fall in the curve is not the result of a change in the volume of the blood. These conclusions are probably correct, for did the fall in the blood-sugar percentage depend on this factor, it would mean that in some cases a dilution of a hundred per cent. or more must take place; for a rapid fall from 0.19 to 0.09 per cent. is frequently seen.

Whatever the fundamental significance of this transient rise in the blood-sugar after the ingestion of carbohydrate may be, there is a consensus of opinion as to the value of a determination of the shape of the blood-sugar curve, which portrays this rise, in the diagnosis and prognosis of cases of diabetes mellitus. The height and the length of the curve are an index of the severity of the disease. Conditions other than diabetes have been described in which variations of the normal curve may occur. A perusal of the published work shows, however, that in none of these is the variation so gross nor so constant as in advanced diabetes, although some of them may resemble closely the type of curve found in cases of mild diabetes.

Nephritis

Chronic interstitial nephritis is one of the conditions in which hyperglycaemia and a prolonged blood-sugar curve has been frequently reported. The type of curve which has been described in many cases is one with an original fasting blood-sugar level of between 0.12 and 0.16 per cent., mounting slowly after 100 gm. of glucose to reach a height of 0.20 to 0.25 per cent. in from 1 to 1½ hours, and subsiding slowly to regain its original level about 3 hours after the first estimation; a curve closely analogous to that of a mild diabetic. There are, however, many cases of chronic nephritis on record with normal blood-sugar curves. Thus hyperglycaemia cannot be looked upon as a *sine qua non* in this disease.

Tachau examined 5 cases of chronic nephritis and found normal blood-sugar levels in 3 of them; in the other 2 the blood-sugar was still raised above 0.200 per cent. 1 hour after the ingestion of 100 gm. of glucose. Hopkins, using Bang's method, found a moderate hyperglycaemia in many of those cases of nephritis with a low sulphothalein elimination. Some of the 26 cases he investigated had normal blood-sugars. Bailey, using Lewis and Benedict's method, reported a case of chronic interstitial nephritis in which the blood-sugar curve after 75 gm. of glucose rose from 0.15 per cent. to 0.36 per cent., regaining the original level in 4 hours. He noted that hyperglycaemia was greatest in those cases of diabetes which were complicated by chronic renal disease.

Hamman and Hirschmann studied the blood-sugar curves in 6 cases of nephritis. In each of these the hyperglycaemia after 100 gm. of glucose was abnormal, the blood-sugar being still raised after 2½ hours.

There seems to be sufficient evidence for the belief that in many cases of nephritis the reaction to the blood-sugar curve test may simulate that found in mild diabetes, but no discussion of the results found in the work on which this evidence is based would be complete without reference to the experiments reported by de Wesselow. He has shown, by comparing the picric acid method of blood-sugar estimation of Lewis and Benedict with MacLean's method, that the former may give readings 40 to 50 per cent. too high, and that these may be the result of the presence of creatinin in the blood which, under the conditions of the estimation, gives a colour similar to that produced by glucose in the presence of picric acid. Cowie and Parsons have confirmed this work, and state that epinephrin, acetone, diacetic acid, and creatinin, when present in the blood, will vitiate the results given by the Lewis-Benedict method of estimation and its modifications. If this be so, then in those cases of nephritis in which there was retention of creatinin with other nitrogenous substances, the picric acid methods of estimation would show an apparent rather than a real hyperglycaemia.

Diseases of the ductless glands

An application of the blood-sugar curve test to cases in which there was disease of the ductless glands has yielded a few isolated results which are very suggestive. But viewed as a whole the results

are extremely indefinite and contradictory. Croser Griffith has reported a case of the hypopituitarism of Fröhlich with a blood-sugar curve of increased sugar tolerance. One hour after 100 gm. of glucose had been given by the mouth the blood-sugar level was 0.104 per cent. and there was no glycosuria. After 2 months' treatment with pituitary extract sugar tolerance was diminished, and an altered blood-sugar curve resulted. A reading of 0.18 per cent. 1 hour after 100 gm. of glucose was now obtained, and sugar was passed in the urine. Bailey, on the other hand, has reported a case of hypopituitarism with a prolonged hyperglycaemic blood-sugar curve. Janney and Isaacson have reported a case of acromegaly with a prolonged curve of the diabetic type. They also investigated cases of exophthalmic goitre, cretinism, and myxoedema. The results of this work, when grouped with the results reported in a later paper by Janney and Henderson, show that, so far, it has been impossible to demonstrate by the use of the blood-sugar test any constant relation between the functional activity of the thyroid gland and sugar tolerance. Many of the curves both in exophthalmic goitre and in myxoedema were normal.

McCrudden and Sargent hold that there is persistent hypoglycaemia in muscular dystrophy.

Cancer

A number of observations on the blood-sugar levels in cases of cancer and other malignant growths have been reported by workers in America. A high prolonged curve has been described as being present in many cases; but, as in nephritis, a number of cases with normal blood-sugar curves have been recorded. Benedict and Lewis, in an investigation carried out on 53 cases of malignant growth, found that in 10 of these the blood-sugar was still raised 3 hours after a carbohydrate meal.

Fredenwald and Grove, using Epstein's method, claim to have found a characteristic and constant deviation from the normal blood-sugar curve in cancer. In 32 cases of cancer of the gastrointestinal tract they estimated the blood-sugar before a dose of 100 gm. of glucose, and 45 minutes and 120 minutes later. They found a high initial level, which after 45 minutes was 0.24 per cent. or higher, and remained at this level for at least 2 hours, showing no tendency to subside during this time. This they termed the 'cancer curve'. They concluded that the blood-sugar curve test

was a valuable aid to diagnosis in cases of carcinoma. There was wide variation in their actual readings. The fasting blood-sugar levels in the 32 cases lay between 0.120 and 0.384 per cent. In 11 of the cases the blood-sugar rose above 0.250 per cent., and yet only 1 of these showed any glycosuria during that time. This unusual feature would appear to indicate either that the renal threshold for sugar was considerably raised in all these cases, or that the method of estimation tended to record readings that were much too high.

Rohdenburg, Bernhard, and Krehbiel contributed two papers on this subject. In the first their results in 24 cases of carcinoma agreed substantially with those of Fredenwald and Grove, a high prolonged curve of the diabetic type resulting. However, in the second paper they modified their views after finding normal blood-sugar curves in about half the cases investigated. They conclude that there is no type of blood-sugar curve that can be considered as characteristic of cancer, but that hyperglycaemia and a delayed curve are often present in that condition.

Chronic arthritis

Pemberton and Foster, in a study of cases of chronic arthritis occurring in the American Army, noted that the severest cases had a diminished sugar tolerance and showed an abnormal response to the blood-sugar curve test. Using the Benedict modification of the Lewis-Benedict method, they then carried out a routine examination of these cases in the stages of marked severity, of convalescence, and of recovery when such resulted. They found that, when the disease was at its worst, there was usually a hyperglycaemic delayed curve, and that in those cases which were improved or cured by the removal of foci of infection such as septic tonsils and carious teeth, a normal blood-sugar curve was restored. They advance the theory that many of the diabetic type of curves, that have been described in various diseases other than diabetes, may be the result of some unrecognized septic focus.

The response of infants and children to the test has been studied mainly by German workers. Mogwitz, in 6 children between the ages of 4 months and 13 months, found that their fasting blood-sugar levels were the same as those of adults. The blood-sugar curve was determined in these cases after feeding them with milk which provided about 2 gm. of sugar per kilo of body-weight. In

4 of these cases there was but little response, the blood-sugar curve rising at the most 0.02 per cent. after half an hour. The highest points in the rise of the other cases were 0.124 per cent. and 0.134 per cent.

Frank and Mehlhorn experimented on 18 children under the age of 3. They gave to them 8 gm. of saccharose per kilo of body-weight. In 8 of the patients glycosuria resulted; in 2 of these the renal thresholds were at 0.130 and 0.160 per cent., which are considerably below those usually given for an adult.

Bergmark investigated the response of the blood-sugar to the ingestion of various sugars in infants. He claims that saccharose produced a higher response than maltose and maltose a higher response than lactose. But all the resultant curves were much lower than those of adults. The average maximal response to doses of glucose in infants under 12 months was a rise to 0.130 per cent.

METHODS

Most of the experiments recorded in this paper were carried out 5 to 6 hours after a light breakfast. This, in the normal individual, is a sufficient period of fasting to allow the influence of the previous meal to pass off. The amount of carbohydrate given varied according to the age of the patient. To adults 50 gm. of glucose dissolved in 200 c.c. of water was usually given. This is sufficient, as MacLean and de Wesselow have shown, to provoke the maximal response, and larger doses need not be given. The amount given to the infants was roughly 2 gm. of sugar per kilo of body-weight. In older children the proportion was less.

MacLean's method of blood-sugar estimation was used throughout. The advantages of this method are well known. Capillary blood is collected in a 0.2 c.c. pipette from a prick in the finger in adults, or from the lobe of the ear or the heel in children. Thus the necessity for venous punctures, which many of the other methods entail, is avoided, and blood can be obtained with great frequency with the minimum of discomfort to the patient. The experimental error was very slight, as will be seen from the following figures.

These are duplicate readings in the case of a man who was given 50 gm. of glucose. Both pipettes were filled simultaneously from the same drop of blood.

	(1)	(2)
Blood-sugar before glucose (1 p.m.) =	0.088 per cent.	0.090 per cent.
„ $\frac{1}{2}$ hour later (1.30 p.m.) =	0.213 „	0.210 „
„ 1 hour later (2 p.m.) =	0.190 „	0.190 „

The accuracy of the method and the constancy of a healthy person's blood-sugar level under fixed conditions are illustrated in the following figures.

On eight days extending over a period of 3 weeks, blood was taken from the same individual at the same hour in each day. In each case it was 4 hours after a light breakfast. The results were:

22.12.20 =	0.089 per cent.
30.12.20 =	0.095 „
31.12.20 =	0.098 „
3.1.21 =	0.091 „
6.1.21 =	0.092 „
10.1.21 =	0.098 „
12.1.21 =	0.096 „
14.1.21 =	0.089 „

MacLean's method has proved so satisfactory in our hands that Cole's modification of it has not been used. This modification, for which the author in the sixth edition of his textbook (1920) appropriates the name 'Cole's method of blood-sugar estimation', is slight, and, as he admits, the principle of the original method is retained. Cole's so-called 'modification' does not appear to be any improvement on the original method as described by MacLean; in fact it is difficult to see wherein the necessity arose to replace the original simple and accurate method by his more or less cumbersome changes, which, without enhancing the delicacy of the method, have rendered it much more difficult to carry out.

THE BLOOD-SUGAR CURVE IN HEALTHY YOUNG ADULTS

In Fig. 6 the normal curve of a young adult is given. This curve represents the average of results which were obtained from 6 healthy men between the ages of 25 and 39, following the ingestion of 50 gm. of glucose. This confirms the findings of many of the authors whom we have quoted. The rise is rapid to a level of about 0.17 per cent., and the return to the original level is complete within $1\frac{1}{2}$ hours. We shall use this as a basis for comparison with

other curves to be described. The highest and the lowest curves which were obtained in this series of 6 cases are also given. They illustrate the point that, whereas the actual height attained by the curve in the normal person is variable, the time required for the return to the original level is more constant.

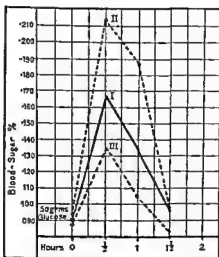


FIG. 6. Blood-sugar curves of normal young adults after the ingestion of 50 gm. of glucose.

I = average of 6 cases. II = highest curve found.
III = lowest curve found.

THE BLOOD-SUGAR CURVE IN CHILDREN

The results obtained in an investigation of the curves in infants under 12 months of age are given in Table 5, and represented graphically in Fig. 7. The amount of carbohydrate given was considered to be equal to the amount the infant would receive in an ordinary feed, and thus sufficient to provoke the maximum response in the blood-sugar. Here again, the average of the results of all the experiments in this group has been taken, and the curves represent the average and the highest and lowest findings of the series. A comparison of these with the normal adult curve reveals a marked difference. As a general rule it appears that the younger the infant the less the hyperglycaemia following the ingestion of sugar. This would suggest how active the carbohydrate storage

mechanism is in very young children. The rate at which the infant is fed must be taken into account, for if the milk or sugar solution were ingested slowly over a period of 20 to 30 minutes, the rate of

TABLE 5
The blood-sugar curve in infants under 12 months

Name	Age	Weight in kilos.	Amount of sugar taken	Before taking sugar	Blood-sugar %		
					$\frac{1}{2}$ hour after	1 hour after	1½ hours after
B. D.	6 days	4	80 c.c. of breast milk = 5 gm. lactose	0.088	0.112	0.103	0.089
L. G.	2 weeks	3½	7.5 gm. lactose	0.080	0.094	0.110	0.098
C. B.	6 weeks	3	10 gm. glucose	0.094	0.140	0.139	0.112
—	10 weeks	4½	7.5 gm. glucose	0.098	0.110	0.105	0.090
S. R.	4 months	6	100 c.c. cow's milk + lactose = 9 gm. lactose	0.092	0.097	0.101	0.097
J. C.	8 months	7½	15 gm. saccharose	0.098	0.117	0.092	0.103
"	"	"	15 gm. lactose	0.112	0.093	0.160	0.096
A. D.	8 months	8	8 gm. lactose	0.108	0.127	0.149	0.098
"	"	"	10 gm. glucose	0.098	0.114	0.107	0.097

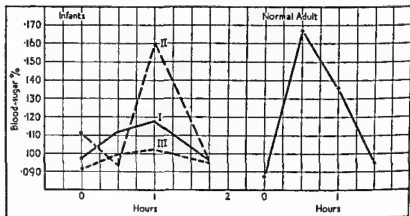


FIG. 7. Comparing blood-sugar curves of infants with a normal adult curve.

I = average of curves of 9 infants. II = highest curve found. III = lowest curve found.

absorption might never be great enough to effect a sudden rise in the blood-sugar. But in these cases attention was paid to this point. The sugar solution was swallowed rapidly in from 5 to 10 minutes.

In two of the cases (J. C. and A. D.) lactose produced a more pronounced rise than saccharose and glucose, both of which appear to be absorbed and assimilated readily.

TABLE 6

The blood-sugar curve in children between the ages of 2 years and 7 years

Name	Age in years	Amount of sugar taken	Before taking sugar	Blood-sugar %			Remarks
				$\frac{1}{2}$ hr. after	1 hr. after	1½ hrs. after	
A. G.	4	15 gm. glucose	0.106	0.187	0.131	0.100	Healthy
B.	4	20 gm. glucose	0.094	0.150	0.106	..	"
R. S.	2	15 gm. glucose	0.105	0.141	0.135	0.090	"
"	"	15 gm. lactose	0.094	0.122	0.103	..	"
C. S.	3	20 gm. saccharose	0.097	0.152	0.103	0.093	"
J. T.	7	Carbohydrate meal = 35 gm. sugar	0.111	0.134	0.114	0.080	"
C. D.	2	15 gm. glucose	0.083	0.155	0.161	0.140	A severe case of active rickets
A. W.	6	Carbohydrate meal = 25 gm. sugar	0.089	0.134	0.219	0.170	A case of encephalitis lethargica with occasional glycosuria
H. S. C.	7	20 gm. glucose	0.112	0.222	0.117	..	A case of muscular dystrophy
E. F.	7	25 gm. glucose	0.103	0.190	0.212	0.260	A case of diabetes mellitus

In Table 6 the results obtained in children between the ages of 2 years and 7 years are given. The response in these is much greater than that which was found in infants; and in children above the age of 3 years it appears to be similar to that found in young adults. The normal adult curve can be taken, therefore, as representing the rise which occurs in all children above that age, and any variation from this would indicate an alteration in carbohydrate tolerance.

The results found in the cases of rickets, encephalitis, muscular dystrophy, and diabetes are also given. The case of diabetes was a boy aged 7 (E. F.), whose blood-sugar had been reduced to a normal level by a diet of limited carbohydrate. A typical diabetic curve followed the ingestion of 25 gm. of glucose. The case of rickets (C. D.), and the case of encephalitis lethargica (A. W.), had a diminished carbohydrate tolerance. In both the curve was prolonged, the blood-sugar being raised considerably 1½ hours after the dose of carbohydrate had been taken.

THE BLOOD-SUGAR CURVE IN OLD AGE

In Table 7 the results obtained in 5 old men over 60 years of age are given. All of them were apparently healthy, with normal cardio

TABLE 7
Blood-sugar curves in old age

Name	Age	Amount of sugar taken	Before taking sugar	Blood-sugar %				Remarks
				$\frac{1}{2}$ hr. after	1 hr. after	1½ hrs. after	2 hrs. after	
N. B.	62	50 gm. glucose	0.138	0.177	0.212	0.219	0.201	Dyspepsia
R. L.	77	" "	0.149	0.197	0.168	0.125	0.109	Healthy
W. G.	74	" "	0.180	0.225	0.230	0.125	0.098	Healthy
								Convalescent after minor operation
W. B.	75	" "	0.117	0.210	0.188	0.148	0.156	Healthy
								Convalescent after minor operation
J. L.	71	" "	0.149	0.160	0.198	0.170	0.162	Healthy

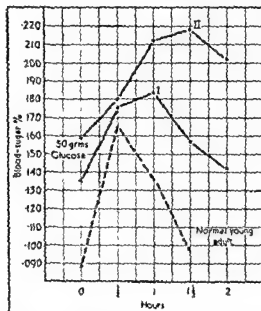


FIG. 8. Blood-sugar curves in old age following the ingestion of 50 gm. of glucose.

I = average of curves found in 5 old men. II = highest curve found (no glycosuria resulted in this case).

vascular and renal systems. In each case the experiment was carried out after 5 hours' fasting; and a dose of 50 gm. of glucose was given. In Fig. 8 the curve of the average of these results is shown

and compared with the normal adult curve. The persistent hyperglycaemia and diminished carbohydrate tolerance of four of these cases (N. B., R. L., W. B., J. L.) is striking, and shows how the carbohydrate storage mechanism tends to become impaired as age advances.

It is significant that the only cases which had a normal fasting level were 2 (W. G., W. B.), who were in hospital convalescing from minor operations, and on a limited carbohydrate diet. The other 3, with no restriction of their diet, appeared to be living in a state of persistent hyperglycaemia. Since this is a condition that can be easily corrected in most cases by a slight modification of the diet, its early recognition by means of blood-sugar estimations would be of value. Rational advice as to the mode of life to be pursued could then be given.

THE BLOOD-SUGAR CURVE IN CANCER

Four cases of cancer were investigated, and the results are given in Table 8. It will be seen that in one of these (C. B.) there is a marked diminution in carbohydrate tolerance. The curve is prolonged and resembles that found in mild diabetics. Two of the cases (B. W., N. N.) present normal blood-sugar curves.

TABLE 8
Blood-sugar curves in cancer

Name	Age	Amount of sugar taken	Before taking sugar	Blood-sugar %				Remarks
				$\frac{1}{2}$ hr. after	1 hr. after	1½ hrs. after	2 hrs. after	
C. B.	54	50 gm. glucose	0.154	0.172	0.175	0.213	0.197	A case of carcinoma of colon with secondary deposits in the liver
G. W.	64	" "	0.102	0.144	0.180	0.133	0.089	Carcinoma of colon
B. W.	30	" "	0.092	0.153	0.130	0.104	..	Carcinoma of colon (colectomy)
N. N.	38	" "	0.093	0.176	0.120	0.102	..	Carcinoma of hp

Thus we have been unable to confirm the findings of Fredenwald and Grove, who maintain that a specific type of delayed

THE BLOOD-SUGAR CURVE IN OLD AGE

In Table 7 the results obtained in 5 old men over 60 years of age are given. All of them were apparently healthy, with normal cardio-

TABLE 7
Blood-sugar curves in old age

Name	Age	Amount of sugar taken	Before taking sugar	Blood-sugar %				Remarks
				$\frac{1}{2}$ hr. after	1 hr. after	1½ hrs. after	2 hrs. after	
N. B.	62	50 gm. glucose	0.158	0.177	0.212	0.219	0.201	Dyspepsia
R. L.	77	" "	0.149	0.197	0.168	0.125	0.109	Healthy
W. G.	74	" "	0.110	0.125	0.130	0.125	0.098	Healthy
								Convalescent after minor operation
W. B.	75	" "	0.117	0.210	0.188	0.148	0.156	Healthy
								Convalescent after minor operation
J. L.	71	" "	0.149	0.160	0.198	0.170	0.162	Healthy

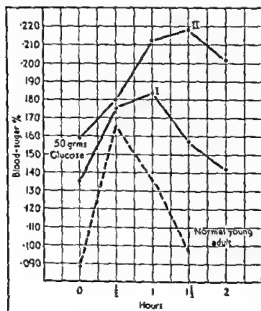


FIG. 8. Blood-sugar curves in old age following the ingestion of 50 gm. of glucose.

I = average of curves found in 5 old men. II = highest curve found (no glycosuria resulted in this case).

vascular and renal systems. In each case the experiment was carried out after 5 hours' fasting; and a dose of 50 gm. of glucose was given. In Fig. 8 the curve of the average of these results is shown

- BING, H. J., and JAKOBSEN, B. 1914. *Dtsch. Arch. klin. Med.* 113, 1913-14, p. 571.
- COLE, S. W. 1920. *Practical Physiological Chemistry*, 6th ed., Cambridge, Cambridge University Press, 1920, p. 253.
- COWIE, D. M., and PARSONS, J. P. 1920. *Arch. intern. Med.* 26, 1920, p. 333.
- DE WESSELOW, O. L. V. 1919. *Biochem. J.* 13, 1919, p. 148.
- FISHER, G., and WISHART, M. B. 1912-13. *J. biol. chem.* 13, 1912-13, p. 49.
- FRANK, A., and MEHLHORN, L. 1920. *Jb. Kinderheilk.* 91, 1920, p. 313.
- FRANK, E. 1910-11. *Z. physiol. Chem.* 70, 1910-11, p. 291.
- FREDENWALD, J., and GROVE, G. H. 1920. *Amer. J. med. Sci.* 160, 1920, p. 313.
- GOTO, K. 1918. *Arch. intern. Med.* 22, 1918, p. 96.
- GRAHAM, G. 1916-17. *Quart. J. Med.* 10, 1916-17, p. 245.
- GRIFFITH, J. P. C. 1918. *Amer. J. Dis. Child.* 16, 1918, p. 103.
- HANDMAN, L. 1919. *Contributions to Medical and Biological Research Offered to Sir William Osler*, New York, P. Hoeber, 1919, p. 845.
- and HIRSCHMANN, I. T. 1917. *Arch. intern. Med.* 20, 1917, p. 761.
- HOPKINS, A. H. 1915. *Amer. J. med. Sci.* 149, 1915, p. 254.
- JACOBSEN, A. T. B. 1913. *Biochem. Z.* 56, 1913, p. 471.
- JANNEY, N. W., and HENDERSON, H. E. 1920. *Arch. intern. Med.* 26, 1920, p. 297.
- and ISAACSON, V. 1918. *Ibid.* 22, 1918, p. 160.
- MCCRUDEN, F. H., and SARGENT, C. S. 1916. *Ibid.* 17, 1916, p. 465.
- MACLEAN, H. 1919. *Biochem. J.* 13, 1919, p. 135.
- and DE WESSELOW, O. L. V. 1920-1. *Quart. J. Med.* 14, 1920-1, p. 103.
- MOGWITZ, A. G. 1914. *Mtschr. Kinderheilk.* 12, 1914, p. 569.
- PENBERTON, R., and FOSTER, G. L. 1920. *Arch. intern. Med.* 25, 1920, p. 243.
- ROHDENBURG, G. L. 1920. *Amer. J. med. Sci.* 159, 1920, p. 577.
- and others. 1919. *J. Amer. med. Ass.* 72, 1919, p. 1528.
- ROLLY, Fr., and OFFERMAN, Fr. 1913. *Biochem. Z.* 49, 1913, p. 278.
- SAKAGUCHI, K. 1918. *Mitt. med. Fak. Tokio*, 20, 1918, p. 345.
- STROUSE, S. 1920. *Arch. intern. Med.* 26, 1920, p. 751.
- TACHAU, H. 1911. *Dtsch. Arch. klin. Med.* 104, 1911, p. 437.

IX. Clinical Tests of the Antirachitic Activity of Calciferol¹

THE pure vitamin D in crystalline form was isolated simultaneously by workers in England, who gave it the name 'calciferol', and by workers in Germany, who called it 'vitamin D₂'. The researches leading to its isolation, with the experimental proof of its antirachitic activity and the measurement of the intensity of this action, are recorded in the Medical Research Council's report on the vitamins (1932). The therapeutic action of calciferol was demonstrated by its effect on experimental rickets in rats, and quantitative determinations carried out on these animals, in comparison with the International Standard Preparation, showed that the pure substance has an activity of about 40,000 international units per milligramme, an ordinary good sample of cod-liver oil showing, in a similar quantitative test, an activity of about 100 units per gramme. It was important to know whether this experimental determination of the activity of calciferol in the accepted units would hold good also for the therapeutic action of the pure vitamin in the treatment of human rickets. The Therapeutic Trials Committee of the Medical Research Council accordingly invited me to carry out clinical trials on calciferol, and this report describes the result of these.

The calciferol was manufactured by the British Drug Houses, Ltd., who supplied it as Radiostol, an oil solution containing 7.5 mg. of pure crystalline calciferol per 100 c.cm., equivalent to 3,000 units per c.cm.

CHOICE OF SUITABLE CASES OF ACTIVE RICKETS

The investigation was started in February 1932. From various sources young children with active rickets were collected. These were then subjected to a clinical and radiographic examination, and suitable cases were chosen and induced to attend at a special clinic. Care was taken to choose uncomplicated cases of active

¹ Published in the *Lancet*, 225, 1933, p. 911.

rickets free from nephritis, urinary infection, coeliac disease, and other chronic disorders likely to interfere with absorption and digestion. It was planned that the majority of these cases would be kept under observation and treatment as out-patients, so the choice of suitable cases was determined by two factors: first, the radiographic evidence of active rickets at the radial and ulnar epiphyses of the right wrist; and, second, the fixed social and economic condition of the family from which the child came. This second factor involved careful inquiry into the income, housing, and dieting of the family; and by intimate questioning week by week or by house visiting when necessary, it was possible to confirm that there was no substantial variation in the basal diet of the children during the course of the investigation. Since all of the families were in receipt of poor-law relief, with only 10 to 20 shillings a week available for food for all the members, it would have been difficult for any important alteration or fluctuation in the diet or hygiene to have taken place without being discovered. To reduce the personal error to a minimum, all clinical examinations and taking of histories were carried out by myself week by week.

In this manner 44 children with active and radiologically proved rickets were taken under supervision—either as controls or for treatment. Of these 19 were rejected during the course of the investigation, either because it was reported by the mother that they had received gifts of food or because the fathers had found employment, under which circumstances it was assumed that the basal conditions of diet would alter and improve. Of the remaining 25 cases I was satisfied—as far as is possible in clinical observation—that the cases were suitable for my purpose.

Of these 25 cases, 3 were chosen, aged 11 months, 2½, and 5 years, to serve as a control standard of the optimal rate of cure. They were put under good hygienic conditions and given an adequate antirachitic diet containing milk, meat, liver, eggs, butter, and vegetables, with 1 oz. of cod-liver oil daily in 2 cases, and 4 tablets of Radiostol (B.D.H.) daily in the other. Serial radiographic examinations of the wrists were made at weekly intervals, and the results of these were set up as a standard scale, described as 'the scale of optimal cure', which was used as a basis of comparison during the investigation. Concurrently, 12 cases were kept under observation and supervision while remaining in their own homes receiving from 1 to 3 c.cm. daily of the oily solution of calciferol,

but without any alteration in their diet so far as could reasonably be determined. These also were examined clinically and radiologically at regular intervals. This group included 2 cases of adolescent rickets: a boy aged 18 and a girl aged 13.

As the investigation continued through the summer months and into the next winter, and some of the cases came first under observation at a time when automatic cure of the rickets might possibly have been taking place through exposure to sunlight, an attempt was made to control this factor by leaving a further 8 cases for initial periods of from 5 to 19 weeks without antirachitic treatment. After this period of observation and control 6 of these were given calciferol, and any initiation of cure where none had previously been present, or acceleration of it if it had already set in, was noted.

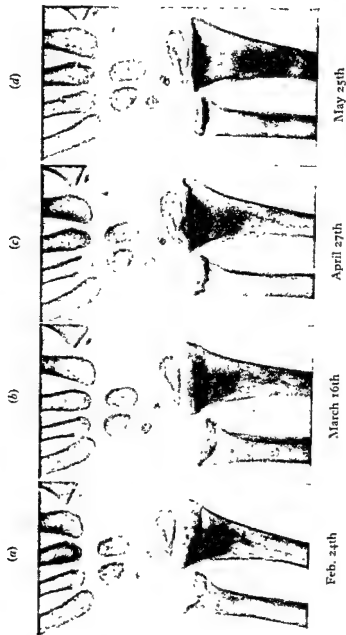
Two pairs of twins with active rickets were observed, one of each pair being used for control.

The remaining case was one which afforded a carefully controlled opportunity of observing the therapeutic effect of calciferol. The child of a negro mother and white father, with active rickets and no radiological evidence of healing, he was in bed indoors for 12 weeks, and it was possible by continuous supervision to be certain that he did not receive other than the diet on which he had developed rickets and which contained no vitamin D. In addition he was treated only with calciferol, and a series of weekly radiographic examinations of the rachitic wrist was made.

Throughout the investigation the degree of rickets in each case was estimated by means of radiographic examination of the right wrist, under a standard technique of radiography arranged by Dr. S. Whately Davidson. The estimation of the rate and degree of progress and cure was by observation of the changes in the serial radiographic pictures.

RADIOGRAPHIC SCALE OF OPTIMAL CURE

The radiological appearance of normal bones (Fig. 9a), of active rickets, and the different stages of healing at the wrists are well known. When the disease is active the ends of the radius and ulna are splayed; their edges are indefinite and lack density; the gap between the ends of the bones and their epiphyses, if they be present, is wider than normal (Fig. 9b). By serial radiographs



Series of radiographs from a child, aged 2 years, with rickets treated with 1 c.cm. calciferol daily. The stages of healing occur at the same rate as in the standard scale of optimal cure. (a) Before treatment. Wide gap (osteoid zone) between shaft of radius and epiphysis. (b) After three weeks. Healing line visible in osteoid zone. (c) After nine weeks. Healing lines consolidating with shafts of bones. (d) After thirteen weeks

taken at weekly intervals it is possible to arrive at a standard scale of normal or optimal cure, noting the time required for the bones to reach specified stages in the healing process, and the time required for ultimate cure. This method does not allow of a directly quantitative expression of the healing process and of vitamin-D efficiency, as does the method used for rats as suggested by R. B. Bourdillon and his associates (Med. Research Council, Spec. Rep. Ser. No. 158, 1931), but it does give a clear indication of the



FIG. 9. The stages of healing of rickets at the ends of radius and ulna in a two-year-old child. (a) Normal. (b) Active rickets. (c) Appearance of first healing line in the osteoid zone between the ends of bones and epiphyses. (d) Organization between the healing line and diaphyses. (e) Dense consolidation of the line with the diaphyses.

presence or absence of a healing response to treatment, and of a comparative rate of cure. The most important stages of the healing process are: (1) the date of the appearance of the first healing line of calcification in the area of the osteoid tissue between the diaphysis and the epiphysis (Fig. 9c); (2) the date of organization of this line with the diaphysis (Fig. 9d); (3) the date of the consolidation of the line with the diaphysis (Fig. 9e). This may be considered the healed stage, although the density of the line of calcification will reveal that the bones have recently been actively rachitic.

The standard of the optimal rate of cure used in this investigation was reached by the observation of the three controls used for this purpose. The results in these confirmed previous observations of my own and other workers, and from these the following *standard of the optimal rate of cure* was set up:

1. The first healing line of calcification appears in the 'osteoid zone' at 2 weeks and not later than 3 weeks.
2. Organization of this line with the diaphysis—i.e., the calcification of the gap between the first healing line and the diaphysis—is visible at 4 weeks and not later than 5 weeks.
3. The consolidation of the line with the diaphysis in a dense firm zone of calcification is visible at 9 weeks and not later than

12 weeks, and at this stage the width of the gap between the diaphysis and the epiphysis is normal.

The use of symbols — and = for the appearance of the first line, \times , $\times \times$, and $\times \times \times$ for organization, and +, ++, and +++ for consolidation, will serve to represent these in graphic form (see Table 9).

One important error may arise in interpreting the radiographs. The healing process of calcification, although described as a line, is in reality a circular rim of a plane surface. It is seen only as a line when this surface is photographed and viewed in one plane. An oblique view may give the appearance of two lines or of a line of lesser density than when viewed in one plane, and this may be misinterpreted as denoting a decrease in the density of calcification.

RESULTS OF CALCIFEROL IN FIRST GROUP

This group was made up of 12 children, 10 of them between the ages of 1 and 5 years, the remaining 2 being cases of adolescent rickets. They were all from poor and badly housed families, averaging three to a room. The typical diet was of bread, jam, margarine, tea, potatoes, and suet puddings, with a little meat once a week. At each attendance a specified dose of calciferol for each child was given in a bottle marked in such a way that the right dose could be accurately gauged. Four of these children were given 1 c.cm. (3,000 units) of the calciferol solution daily, 6 of them 2 c.cm., 2 of them 3 c.cm. daily throughout the period of observation. The remaining case (No. 13) was given 2 c.cm. for 14 days and then the calciferol was replaced for the next 13 weeks by an inert 'bottle of medicine' free from vitamin D. In 5 of the cases treatment was started in February at a season when there was little or no sunshine. The results are shown symbolically in Table 9, and can be summarized as follows. In all the 12 cases which received calciferol continuously satisfactory healing of the rickets took place within 13 weeks—that is, at a rate and within a period equal to the optimal rate of healing of the control group. In all cases an evident healing line was seen within 2 weeks of the start of the period. In only 1 of the 12 cases was the healing line delayed until the fourth week, and in this case healing continued, and at the twelfth week the healing stage was reached.

TABLE 9
The treatment and observation of human rickets

	1	2	3	4	5	6	7	8	9	10	11	12	13	15
Optimal healing in controls	o	—	=	x	x	x	+	+	+	+	+	..
(1) 2 years, Feb., 2 c.cm. daily.	o	?	=	x	x	x	+	..	+	+	+	..
(2) 2 years, Feb., 1 c.cm. daily.	o	—	=	x	x	x	+	..	+	+	+	..
(3) 2 years, Feb., 2 c.cm. daily.	o	—	=	x	x	x	x	..	+	+	+	+	+	..
(4) 1½ years, Feb., 1 c.cm. daily.	o	—	=	x	x	x	x	..	+	+	+	+	+	..
(5) 3 years, Mar., 2 c.cm. daily.	?	—	=	..	x	..	x	+	..	+	+	..
(6) 5 years, Mar., 3 c.cm. daily.	o	—	=	..	x	x	x	..	+	+	..	+	+	..
(7) 1 year, Apr., 1 c.cm. daily.	o	..	=	..	x	x	+	+	+	+	+	..
(8) 2 years, Apr., 1 c.cm. daily.	o	o	=	..	=	x	+	..	+	+	+	..
(9) 1½ years, Apr., 2 c.cm. daily.	o	o	—	..	x	..	x	x	+	..	+	+	+	..
(10) 2 years, Sept., 2 c.cm. daily.	o	—	=	..	x	..	x	x	+	..	+	+	+	..
(11) 18 years, Feb., 3 c.cm. daily.	o	=	=	x	+	+	+	..
(12) 13 years, June, 2 c.cm. daily.	o	..	=	x	+	+	+	+	..
(13) 1½ yrs., Mar., 2 c.cm. for 14 days only.	?	—	..	x	x	x	..	*	..	+	..	+	+	*

In this table the symbols denote X-ray examinations of the wrist and the degree of rickets or stage and degree of healing revealed thereby. The symbol o denotes active rickets without sign of healing; and =, the first healing line; and +, the organization of this line with the diaphysis; and + the consolidation of this line in a dense line of calcification; the symbol ++ denotes the final degree of healing. The symbol * in Case 13 denotes unsatisfactory healing and relapse of rickets. The first column shows the age of the child, the month in which treatment was started, and the amount of calciferol solution given.

The method did not permit of an accurate quantitative estimation of the comparative effects of different amounts of calciferol, but in 3 cases (Nos. 2, 4, and 7 in Table 9) 1 c.cm. of the solution (3,000 units) daily cured the rickets at an optimal rate, the first line of calcification appearing within 3 weeks, and complete healing with dense calcification within 11 weeks. The remaining case (No. 13), which received calciferol only for 2 weeks, showed a satisfactory healing line at 2 weeks. Under this impetus the healing process continued for a further 6 weeks—there was then some apparent retrogression—and at the fifteenth week active rickets was reappearing.

The two cases of adolescent rickets healed satisfactorily with calciferol. One of these was a boy, aged 18, who for some unaccountable reason had begun to develop evident rickets at the age of 15. Prior to that he had apparently been an active healthy boy. Two years earlier the enlargement of the epiphyses at the wrists had become visible, and difficulty in walking commenced. At the time he first came under treatment in February 1932, he was severely affected and able to walk only with a slow waddling gait. To external examination there appeared to be gross active rickets with genu valgum. Radiographs confirmed this, with typical appearances at the radial and ulnar epiphyses without any trace of a healing line. The blood calcium was 8.9 mg. per 100 c.cm. of serum; phosphorus, 1.72 mg. per 100 c.cm. of plasma; blood urea, 26 mg. per 100 c.cm. No albumin in the urine. There was no history of diarrhoea and no evidence of coeliac disease. The Wassermann reaction was negative. On inquiry his diet appeared to be satisfactory, but in spite of this the rickets was active and increasing. He was given 3 c.cm. of calciferol solution (9,000 units) daily. Two weeks later a very definite healing line was present in the osteoid tissue beyond both the ulna and the radius. In 3 weeks this was still more evident. He was then unable to attend the clinic until the eleventh week, but the calciferol was taken to him at home, and at the eleventh week there was almost complete healing of the optimal degree.

CASES RECEIVING NO CALCIFEROL

At various stages during the investigation 8 cases were set aside at their first attendance to be kept under observation during a

period when they received no calciferol or other vitamin D preparation. This was done to estimate and control the rate and degree of automatic healing which might take place, particularly during the summer months. The period of observation varied from 19 to 5 weeks in the individual cases. In some of these there were signs of slight but slow healing, but this occurred only between the months of May and September, and in none of them did it take place at the optimal healing rate or at the rate of those treated with calciferol. Between the months of February and May, and again after October, the rachitic bones in this group of children showed no healing, and in 1 case there was an actual increase in the degree of rickets observed during November and December. When automatic healing did take place during the summer months it was in a different and less efficient manner than in the cases treated with calciferol. The initial healing processes in the osteoid tissue were in scattered points and did not appear in a definite line. Consolidation in a dense calcification was absent in all but one case. The healing process fluctuated and was not steadily progressive. In only 1 of these cases did the healing approach the standard rate of normal optimal healing and that was in a negro child, aged 4, who came under observation in the month of May. The details of these cases are as follows:

CASE A. Aged 2½ years. Seen for 10 weeks from February to May without calciferol. On 22 February 1932, active rickets; no healing. At 6 weeks rickets more marked; at 8 weeks doubtful evidence of healing process; at 10 weeks definite but slight evidence of healing, but not equal to that of the third week in the standard scale of optimal rate of cure.

CASE B. Aged 3 years. Seen for 8 weeks from May to July without calciferol. On 9 May 1932 florid active rickets, no healing. At 3 weeks no healing. At 5 weeks earliest evidence of slight healing; at 7 weeks no change; at 8 weeks healing progressing but not equal to that of the fourth week in the standard scale. No organization or consolidation of healing calcifying centres.

CASE C. A negro boy, aged 4. Seen for 9 weeks from May to July without calciferol. On 18 May 1932 florid active rickets, no healing. At 3 weeks no healing; at 6 weeks healing evident; at 9 weeks definite and almost satisfactory healing, but line not as dense or consolidated as in standard scale.

CASE D. A twin boy, aged 6. Seen for 19 weeks from June to October without calciferol. On 9 June 1932 gross florid active rickets. At 2 weeks no healing; at 4 weeks earliest evidence of scattered healing; no line; at 5 weeks no change; at 7 weeks healing equal in degree to third week of standard scale; at 13 weeks still evident active rickets, but centres of healing in osteoid tissue without consolidation; at 16 weeks slow irregular healing; at 19 weeks rickets not yet healed, wavy slightly calcified outline to ends of bones—not equal to seventh week of standard scale of cure.

CASE E. Aged 6, twin brother of Case D. Seen without calciferol for 5 weeks from June to July. At 5 weeks only slight evidence of inefficient healing, not equal to the second or third week of standard scale of cure. (Treated subsequently with calciferol.)

CASE F. Aged 2½. Seen for 11 weeks from June to September without calciferol. On 20 June 1932 active florid rickets. No healing in first 4 weeks. At 11 weeks rickets still active, but slight evidence of healing though not equivalent to third to fourth week of standard scale of cure.

CASE G. Aged 4, a twin boy. Seen for 18 weeks from June to October without calciferol. On 1 June 1932 gross florid rickets without healing. At 4 and 5 weeks slightest degree of early healing; at 8 weeks, healing progressing but less than at fifth week of standard scale; at 14 weeks rickets still active; at 16 and at 18 weeks slow, irregular calcification, but rickets not yet healed, equivalent to seventh week of standard scale.

CASE H. Aged 2½. Seen for 5 weeks from November to December without calciferol. On 28 November 1932 florid active rickets, no healing. At 3 weeks rickets worse; at 5 weeks rickets worse, no healing.

In summary, the results in this control group of 8 cases without calciferol or other vitamin D treatment were: In 1 case, a negro boy (C), automatic healing took place in the months of May to July, at a rate almost equivalent to the standard optimal rate of cure; in 1 case (H) seen for 5 weeks during the months of November and December there was absolutely no healing; in the remaining 6 cases, slow improvement took place between the months of May and September, but not at the standard optimal rate, and in 2 of these after 18 and 19 weeks their rickets was still active and uncured.

RICKETS IN TWO PAIRS OF IDENTICAL TWINS

Of two pairs of identical twin boys, one of each was treated with calciferol, the other without any extra source of vitamin D serving as control. One pair of these was Cases D and E of the preceding group.

They were aged 6 years, members of a family of eight children and two parents living in a single basement room. Both had extremely severe florid rickets, with lateral bowing of the tibiae and slow waddling gait. For an initial period of 5 weeks both were treated without calciferol and given a little extra calcium daily. For the next 14 weeks one twin (E) was given 2 c.cm. of the calciferol solution daily; the other twin (D) was given a 'medicine' free from vitamin D.

At the onset on 9 June E (the calciferol-treated twin) had blood calcium of 9.0 gm., and blood phosphorus 3.2 mg. per 100 c.cm. with extreme rickets seen radiologically at the ulnar and radial epiphyses. During the initial 5 weeks without calciferol there was only very slight automatic healing (equal in both twins). From the sixth to the nineteenth week the boy took the calciferol daily. During this time cure took place at the optimal standard rate, and healing was complete at the nineteenth week—i.e. after 13 weeks of calciferol. On 11 October (twelfth week of calciferol) blood calcium was 10.5 mg. and blood phosphorus 5.16 mg. per 100 c.cm.

The other twin (D) was kept under observation for the whole period of 19 weeks without calciferol. On 9 June the blood calcium was 9.2 mg. and blood phosphorus 4.2 mg. per 100 c.cm. He showed very slight automatic healing during the initial period of 5 weeks, but at a slow rate and in an inefficient degree. At the nineteenth week his rickets was still uncured. On 11 October blood calcium was 9.9 mg. and blood phosphorus 3.59 mg. per 100 c.cm.

In this pair of twins the radiological evidence showed that the calciferol (2 c.cm. daily) produced satisfactory healing of the rickets at the optimal rate, compared with the exact control of his brother. The radiological evidence was confirmed by the blood calcium and phosphorus results in both cases, the ultimate levels being 10.5 mg. Ca and 5.1 mg. P in the calciferol-treated case, and 9.9 mg. Ca and 3.59 mg. P in the control case. It was confirmed again by the clinical condition of the children. The calciferol-treated child became more quickly active in his gait.

The other pair of twins were boys aged 4, who came under observation on 1 June with florid active rickets. One of them was treated from the outset with 3 c.cm. of calciferol solution daily. His rickets healed and cured at the optimal rate equivalent to the standard scale and other calciferol cases. No radiographs are available from the eighth to the sixteenth weeks, but at the eighth week there was firm consolidation of the healing line, and firmly healed at the sixteenth week. The other twin (not treated with calciferol) showed slow progressive automatic healing throughout the summer months, but this was not yet complete at the eighteenth week, when the degree of healing was equivalent only to that of the eighth to ninth week of healing of the standard scale of optimal rate of cure.

CASE ON CONTROLLED DIET AND CALCIFEROL

A half-caste boy, aged 2, came under observation on 4 February 1932. The family of five lived in one room. They lived on a typically rachitogenic diet. He had severe active rickets. He could not stand or walk. Radiographs showed an extreme degree of actively rachitic bones, with a very wide gap between the end of the radius and its epiphysis. From February to May he was kept under close observation in bed on a known diet consisting of bread, treacle, tea, sugar, and lemon juice to breakfast; cabbage, potato, one ounce of beef, bread and treacle to dinner; bread, jam, apple, tea, and lemon juice to supper, and extra dry sweet biscuits at night; and treated with 2 c.cm. of the calciferol solution daily. It was possible to be certain from the close supervision which was maintained that his diet contained no known vitamin D other than the calciferol, and also that he was not exposed to sunlight. Serial radiographs showed that the calciferol produced active and rapid healing. After 3 weeks a healing line was visible in the osteoid zone. This was extremely well marked across the width of the bone ends in the fourth week, and was organizing and consolidating in the fifth week. Healing continued, and at the eighth week the rate and degree of the calcification equalled that of the standard scale of optimal cure. After the eighth week progress was not so satisfactory as in cases treated on a combination of full diet with the vitamin D preparations and sunlight, the final healed stage being reached at 14 to 15 weeks

instead of 9 to 12 weeks of the standard scale of optimal cure. The extremely poor diet probably accounted for this terminal delay.

This observation proved that 2 c.cm. of calciferol solution daily had a definite curative effect on the rickets of a child living on a vitamin-D deficient diet. The immediate calcifying effect was equal to that of a standard rate of cure produced by cod-liver oil or irradiated ergosterol.

CONCLUSIONS

Twelve actively rachitic children were treated with daily doses of calciferol for 11 weeks or more. The therapeutic effects were estimated by serial radiographs, comparing them with a standard scale of the optimal rate of cure. A control of 8 rachitic children without calciferol was used to determine what spontaneous or automatic healing might have occurred had no calciferol been given.

The results in these 12 cases showed that calciferol had an active curative effect on the rickets, and that it produced healing at an optimal rate, acting as quickly and effectively as the usual therapeutic doses of cod-liver oil or irradiated ergosterol.

The results of the antirachitic activity of calciferol were confirmed by observations on two pairs of twins, one member of each being kept as control, and also on a child with an exactly controlled diet.

It has not been possible in this investigation to make an exact quantitative comparison between the therapeutic activity of calciferol and that of other vitamin D preparations, because daily doses of less than 1 c.cm. of the calciferol solution (3,000 units) were not given. But it was shown that in 4 cases, 2 of which were treated from February to April, 1 c.cm. of the calciferol solution produced a maximal curative effect.

Experience gained during the course of this investigation suggests that there are sufficient cases of human rickets in the big towns living under conditions which are suitable for comparative therapeutic tests of antirachitic substances. If such investigations were to be carried out it is advisable that the observations should be commenced between the months of November and January.

attenders at welfare clinics. That the groups represented samples from the same social levels and conditions is revealed by the high percentage of unemployed amongst the parents of each: 'Clinic group', 46 unemployed out of 51; 'Domestic group', 44 unemployed out of 54; 'Casual group', 13 unemployed out of 20. Together, these 125 children may be taken as typical examples of those between the ages of 1 and 5 years to be seen in the poorest houses and streets of the city. Collectively, they are referred to in the report as the 'Labouring and artisan families class', or, more briefly, as the 'City children'.

For contrast and comparison records were collected of the heights and weights and illnesses of the 124 children of the better-class families. These were all children of parents engaged in professions or in commerce, living under good conditions in the residential districts of the city. These are referred to in the report as the 'Professional families class'.

The health and nutrition of the 125 children of the Clinic, Domestic, and Casual groups were estimated from the results of a clinical examination and an inquiry into their environment and previous history. Careful records were made of the following points:

1. The birth history of the child.
2. The growth and development prior to examination.
3. The nature and severity of any illnesses.
4. Any medical or institutional treatment.
5. The diet during and after infancy.
6. The family income and expenditure.
7. The parental capacity.
8. The housing conditions.

CLINICAL EXAMINATION

The clinical examination of the children was as full and complete as possible. Knowing that a child may be thin and appear malnourished, but that this may be due to factors other than faulty diet, a careful search was made for the presence of other relevant or complicating factors, of which illnesses resulting from infectious diseases were the most important. Knowing also from the experience of previous investigations that a child may be suffering severely from rickets, xerophthalmia, scurvy, and other diseases of

malnutrition, and yet be 'normal' in height, weight, and appearance, it was recognized that the clinical examination had to consist of something more than measuring, weighing, and looking at the children.

Particular attention was paid to the following points:

1. General condition, judged from the appearance, the activity, the subcutaneous covering, the amount and tone of muscle, and the stance of the child.
2. Weight and height measurements.
3. The presence of rickets from clinical examination and from X-ray photographs of the wrists.
4. Haemoglobin estimations.
5. Development of the teeth.
6. The presence of signs of systemic diseases, particularly infective cervical adenitis, ear infections, splenomegaly, and lung infection.

Microscopic examinations of the urine and tuberculin skin tests, although desirable in an investigation of this sort, were not practicable on this occasion, and were not carried out.

GENERAL CONDITION

The general condition, i.e. the physical condition judged from the child's appearance, by taking into account its development, musculature, amount of subcutaneous fat and stance, is a useful standard for recognizing those who appear to be in perfect health and nutrition, and those who are obviously thin and in poor condition. But for the assessment of children between these extremes it was found to be unreliable. In the beginning I used categories of (1) Very good, (2) Good and satisfactory, (3) Poor, (4) Very poor. Most of the children fell into the categories 2 and 3, but often the results of further clinical examinations belied the appearances on which they had been classified.

Some classified as of good and satisfactory general condition were found, on further examination, to be suffering from defects of health and nutrition. Others looked upon as of poor general condition, appearing thin or flabby, were found to be healthy in every other respect. For this reason classification of the general

condition was carried only so far as to pick out those in the following categories:

1. Good and satisfactory, i.e. healthy looking, well-developed children.
2. Poor and unsatisfactory, i.e. thin, wasted and badly developed children.

The rest, which might have been classified variously as of 'average' or 'moderate' general condition, without setting any significant or definitive value on the terms, were grouped as 'remainder'. Using these standards, the following results were obtained for the 125 City children in the three sample groups:

Good and satisfactory general condition .	41
Poor and unsatisfactory general condition.	25
Remainder	59

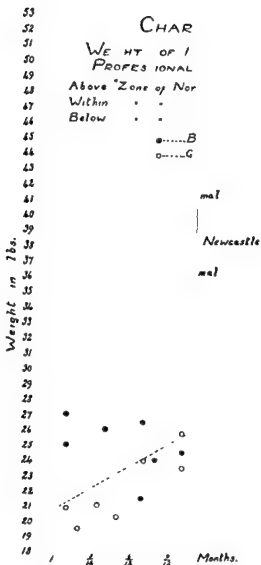
WEIGHT

The weights were measured to the nearest ounce, the children being either naked or clad in one thin under-garment. The results were recorded, and on the accompanying charts are plotted to the nearest quarter-pound. For the purpose of classification and comparison I have used the method of making a normal zone of weight, within the limits of which the child of normal health and development might be expected to fall. The numbers as then classified are:

1. Above the normal zone of weight.
2. Within the normal zone of weight.
3. Below the normal zone of weight.

Plotting the weights on squared paper with the normal zone marked thereon is probably the simplest and most satisfactory method of recording them if they are to be comprehended by the average reader lacking an understanding of higher mathematics. The graphic method reveals how wide is the variation in weight of children at any given age, and how misleading it may be to assess the condition of an individual by a comparison of his weight with an expected 'average' standard.

I constituted the zone of normal weight by using the figures given in Table 10.



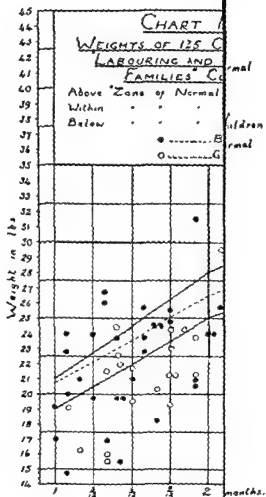


TABLE 10
Zone of normal weight

	<i>At 1 year</i>	<i>At 2 years</i>	<i>At 3 years</i>	<i>At 4 years</i>	<i>At 5 years</i>
Upper limit	21 lb.	28 lb.	33 lb.	37 lb.	41 lb.
Lower limit	19 lb.	25 lb.	29 lb.	33 lb.	36 lb.

Within this normal zone fall most of the normal weight lines in common use (Holt, Bowditch, British Anthropometric Committee, Board of Health). The average weight of girls being less than 4 per cent. below that of boys, the zone is wide enough to be used for both sexes. Within this also falls a line of 'average' weights prepared by the Newcastle upon Tyne Health Department from recent weight figures of 3,966 children of the city clinics, which were as follows:

<i>At 1 year</i>	<i>At 2 years</i>	<i>At 3 years</i>	<i>At 4 years</i>	<i>At 5 years</i>
20½ lb.	26½ lb.	31 lb.	34½ lb.	37½ lb.

This zone of normal weight is shown on Chart No. 1, in which are plotted the weights of 124 children of the 'Professional families class'. These are meant to serve as a basis of comparison, and they reveal that this zone of normal weight is a modest standard to which children may be expected to attain, for 60 of those 'Professional families class' children were above, 48 within, and 16 below its limits.

The results in the three sample groups of City children have been combined, and are shown on Chart No. 2. The following table gives the numerical results of these according to their relation to the zone of normal weight:

TABLE 11

Relation to the zone of normal weight of 3 groups of children

	<i>Above</i>	<i>Within</i>	<i>Below</i>
Group I. Clinic.	7	20	24
Group II. Domestic	7	16	31
Group III. Casual	..	6	14
Total of Labouring and artisan families class	14	42	69

Expressed in percentages, and comparing them with the results of the 'Professional families class', we get the following results:

TABLE 12

Relation to the zone of normal weight of 2 classes of children

	<i>Above %</i>	<i>Within %</i>	<i>Below %</i>
Professional families class (124) . . .	48.4	38.7	12.9
Labouring and artisan families class (i.e. Groups I, II, and III (125) . . .	11.2	33.6	55.2

HEIGHT

The heights were measured to the nearest half-inch, and the results are plotted graphically in Charts 3 and 4. Normal standards of height are represented by a zone, similar to that used in the normal weight standards, and for the construction of this the following figures giving heights in inches were adopted:

TABLE 13

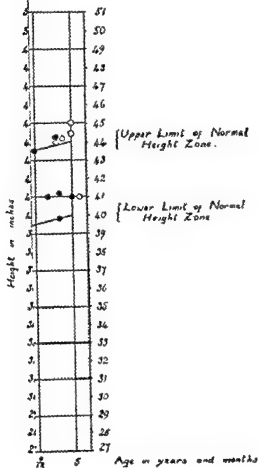
Zone of normal height

	<i>1 year</i>	<i>2 years</i>	<i>3 years</i>	<i>4 years</i>	<i>5 years</i>
Upper limit of normal zone .	30	35	39	42	44
Lower limit of normal zone .	28	32	35	38	40

The standards in common use for the heights of normal children (Holt, British Anthropometric Committee) fall within the limits of this zone, and its lower limit is at a level which at least 80 per cent. of healthy children may be expected to reach (see Chart No. 3). The difference in the 'average' heights of boys and girls at this age is so slight (less than 2 per cent.) that the zone serves for both sexes.

Chart No. 3 shows the heights of the 124 children of the 'Professional families class', and of these 31 (25 per cent.) fell above the zone, 87 (70.2 per cent.) within the zone, and 6 (4.8 per cent.) below the zone.

In the three sample groups of the City children, height measurements were made of 115 of them, with the results shown in Table 14.



	4		49	
	4		48	
	4		47	
	46		46	
	4		45	
			44	{ Upper Limit of Normal Height Zone.
	43	○	43	
	42		42	
	41		41	
	4	•	40	{ Lower Limit of Normal Height Zone
	39		39	
	38	•	38	
	37	○	37	
	36		36	
	35		35	
	3		34	
	33		33	
	32		32	
	31		31	
	30		30	
	29		29	
	28		28	
	27		27	
	26		26	
	25		25	
Height in inches.	1	5	34	Age in years and months

In the sample groups of City children from Labouring and artisan families there were 121 whose haemoglobin values were estimated. The results of these are shown in the following table:

TABLE 17
*Haemoglobin estimations for children from
'Labouring and artisan families'*

<i>Haemoglobin, per cent.:</i>	84-80	79-75	74-70	69-65	64-60	59-55	54-50	<i>Below 50</i>
Number of cases (total 121)	9	15	41	28	13	10	5	..

These results can be better interpreted by expressing them as percentages falling above or below chosen levels.

- (a) Taking the level of 75 per cent. of haemoglobin as that which healthy children should reach, there were:

Above 75% haemoglobin—19·8% of the children.

Below 75% haemoglobin—80·2% of the children.

- (b) Taking the level of 70 per cent. of haemoglobin as a dividing line, there were:

Above 70% haemoglobin—53·7% of the children.

Below 70% haemoglobin—46·3% of the children.

- (c) Taking the level of 65 per cent. of haemoglobin as a dividing line, there were:

Above 65% haemoglobin—76·9% of the children.

Below 65% haemoglobin—23·1% of the children.

For the purpose of defining anaemia no absolute critical level of haemoglobin can be set up. Many children with 70 per cent. of haemoglobin are vigorous and develop well, although that is below the desired or normal value. But it is reasonable to say that children with less than 65 per cent. of haemoglobin should be regarded as anaemic. Using this as a standard for anaemia, and 75 per cent. of haemoglobin as the lower limit of normal, the results of the blood examinations can be summarized as follows:

Of 121 City children from Labouring and artisan families 19·8 per cent. had satisfactory haemoglobin values (above 75 per cent.), and 23·1 per cent. were anaemic (below 65 per cent.). The remainder fell between these standards.

INCIDENCE OF OTHER NUTRITIONAL OR DEFICIENCY DISEASES

In the groups of City children examined no evidences of scurvy, xerophthalmia, nutritional night blindness, nutritional oedema, beri-beri, or pellagra were found. There was a high incidence of respiratory and other infections among them, but how much of the lowered resistance to infection and slow recovery from infective illnesses is to be considered as evidence of improper or inadequate nutrition is difficult to decide. The *immediate* incidence of the common infective illnesses of childhood is certainly more dependant on the factors of age and over-crowding than on nutrition; but it is equally certain that the severity of these illnesses is influenced to some degree by the adequacy or inadequacy of the diet of the children. Leaving aside for the moment the high incidence and severity of various infective illnesses, no signs of actual deficiency diseases other than rickets and anaemia were found in the City children.

INCIDENCE OF ILLNESSES

When the weights and heights of children of the better-class ('Professional families class') were collected, information on the incidence of various diseases amongst them was obtained for the purposes of contrast and comparison. In the same way at the clinical examination of the City children a record was made of the various illnesses and diseases they had passed through. This showed clearly that infection and other diseases were more frequent and occurred at an earlier age in the City children.

Some idea of the relative state of affairs in the two classes will be revealed if I refer to the incidence of respiratory illnesses and measles.

Amongst the 124 better-class children, 2 had suffered from pneumonia, 1 from pleurisy, and 2 others from a chronic and recurrent cough, sufficient in degree to arouse the parents' anxiety and need for treatment.

Amongst the 125 City children, 17 had had pneumonia, and in addition 32 were or had been affected by chronic or recurrent bronchitis.

Only in 6 of the better-class children was there a record of an

attack of measles. Amongst the poor-class children there was a history of measles in 46.

Diarrhoeal diseases no longer play an important part in causing ill health amongst children between the ages of 1 and 5. In only 6 cases was a history of recurrent chronic diarrhoea obtained. On the other hand, a history of abscesses, septic skin infections, and otitis is frequent, and in any further inquiry the significance of these should be studied and taken into consideration.

It will be seen how high is the incidence of infective diseases amongst the City children in their earliest years. This has an important bearing on their physical condition and state of health. The damage that is done by these illnesses, occurring at susceptible ages, and under conditions which prevent satisfactory recovery, accounts for much of the poor physique and ill health in later years. This must be borne in mind in any approach to the problems of malnutrition. It is a factor probably as important as the factors of inadequate diet and improper housing, and intimately connected with them both, for where serious infective disease is found to be the primary cause of permanent physical damage, inadequate housing and improper diet will contribute to its ill effects by providing the opportunity for mass infection, and by lowering resistance to the disease.

SUMMARY OF RESULTS

The results of the examination of the City children recorded above, and the comparison of these with normal standards, reveal the following facts:

- 55.2 per cent. were below the normal standard of weight.
- 47.0 per cent. were below the normal standard of height.
- 23.1 per cent. were anaemic with haemoglobin values below 65 per cent.
- 5 children out of 103 specially examined for the purpose had active rickets.

There was no evidence of other nutritional diseases, such as xerophthalmia, scurvy, or nutritional oedema.

The assessment of the physical condition of the City children, by taking into account only their appearance, stature, and weight, was as follows:

- (a) Good and satisfactory general condition—32.8 per cent.

(b) Poor and unsatisfactory general condition—20·0 per cent.

(c) Remainder ('average' or 'moderate' general condition)—47·2 per cent.

I have suggested, however, that it is not sufficient that a final estimate of the physical condition of children should be made only by observation of their appearance, stature, and weight, and I therefore shall attempt a reclassification of these three groups by taking into consideration the additional information provided by the blood and X-ray examinations, and by a correlation of these factors.

Out of 125 children there were 41 (32·8 per cent.) who were classified by observation of the appearance, stature, and weight as being of 'good and satisfactory general condition'. None of these showed any signs of rickets, but 5 of them were anaemic with haemoglobin values below 65 per cent., and cannot therefore be retained in the category. This leaves 36 children of good and satisfactory appearance and general condition, free from rickets and anaemia.

In the 'remainder' group of 59 children (47·2 per cent.), i.e. the group classified by observation as of 'average' or 'moderate' general condition, there were 3 who had active rickets (1 of whom was anaemic also), and apart from these, there were 12 who were anaemic with haemoglobin values below 65 per cent. These 15 should therefore be transferred and classified in the group of 'poor and unsatisfactory condition'. This leaves 44 children of this 'remainder' group who, although slightly under normal standards of weight and height and general condition, may be looked upon as of satisfactory general condition.

In the group of 25 children (20·0 per cent.) classified by observation as of 'poor and unsatisfactory general condition', all were markedly under normal standards of physical height and weight, 2 had active rickets, and 10 were anaemic. The poor physique of the children of this group, who were free from rickets and anaemia, prevents them being promoted to a higher category, and they remain classified as being of 'poor and unsatisfactory general condition'.

Adding to this last group of 25 children the 3 cases of rickets and the 17 cases of anaemia of the other two groups, the summation of the results is that out of 125 City children examined 45 (36·0 per cent.) were found to be unhealthy or physically unfit.

These 45 'unhealthy or physically unfit' children were distributed in the following age-groups:

Between 1 and 2 years—25 (out of 47 in that age-group—53·2 per cent.).

Between 2 and 3 years—10 (out of 28 in that age-group—35·7 per cent.).

Between 3 and 4 years—7 (out of 29 in that age-group—24·1 per cent.).

Between 4 and 5 years—3 (out of 21 in that age-group—14·3 per cent.).

I have approached the problem of ascertaining how much of the ill health of the children can be attributed to the results of microbic diseases or environment, and how much to improper diet, by an analysis of the histories of the 45 children whom I finally classified in that category.

When the mother gave the history that the child thrived well to a certain age, and then developed an illness and did not thrive afterwards, I have attributed its poor and unsatisfactory condition to the effects of that illness. This sequence of events was clear and unmistakable in many cases, particularly when the illness had been the broncho-pneumonia of measles, otitis media, skin sepsis, and adenitis.

A sample survey was made of the history of 27 healthy children who were of good and satisfactory general condition and free from rickets and anaemia. In 6 of these cases there was a history of illness—3 cases of pneumonia with prompt recovery, and 3 cases of measles with prompt recovery. The remaining 21 children had escaped any serious illnesses up to the time of examination.

The analysis of the 45 'unhealthy or physically unfit' children gave the following results:

There were 20 who gave a history of a preceding illness which I deemed to be the cause of their subsequent condition.

In 6 of these measles or some complication of measles had been the starting-point of their ill health.

In 6 broncho-pneumonia, with slow recovery, had been the starting-point of their ill health.

In 5 otitis media, mastoiditis, or skin sepsis and adenitis had been the starting-point of their ill health.

There remained 25 children of the 'unhealthy or physically

unfit' group who had had no serious illness to which their condition could be attributed. After a careful consideration of the results of their clinical examination and of their diets, I could find no other probable cause than that of improper or inadequate diet, and I feel justified in deducing that the condition of these 25 children was the result of malnutrition.

NOTE ON HOUSING CONDITIONS

A complete analysis of the housing conditions of the families was not made, but the following facts reveal that these conditions probably contribute greatly to the problem of malnutrition and ill health.

(a) The sample of 27 healthy City children of good physique and condition, whose record as regards infectious diseases has previously been referred to, came from families living under the following conditions:

6 families were in 1-roomed tenements or houses with an average of 2.5 persons per room.

7 families were in 2-roomed houses or tenements, with an average of 2.5 persons per room.

10 families were in 3-roomed houses, with an average of 1.3 persons per room.

4 families were in 4-roomed houses, with an average of 1.4 persons per room.

The average of the whole group was 1.7 persons per room.

(b) The 45 'unhealthy or physically unfit' children came from families living under the following conditions:

11 families were in 1-roomed tenements, with an average of 4.1 persons per room.

20 families were in 2-roomed tenements, with an average of 3.1 persons per room.

11 families were in 3-roomed houses, with an average of 2.0 persons per room.

3 families were in 4-roomed houses, with an average of 1.5 persons per room.

The average of the whole group was 2.6 persons per room.

These results denote that there are children who can be brought up in a state of relatively good health in poor housing conditions, where no doubt the fault is compensated by good mothercraft,

efficient housekeeping, and their good fortune in escaping serious illnesses at an early and susceptible age. On the other hand, this examination of the housing conditions shows that there is a direct relationship between overcrowding and ill health or malnutrition. If serious illnesses and improper diet be accepted as the prime causes of these, the overcrowding in bad housing must be looked upon as an important contributory factor, fostering as it does the chance of mass infection, and impairing the efficiency of the parents in their task of providing a proper and adequate diet for their children.

CONCLUSIONS

There are at present no definite standards or formulae by means of which the physique and state of nutrition of children can be estimated with mathematical accuracy. An expression of the incidence of poor physique or malnutrition must therefore be regarded as an opinion, the validity of which will vary with the experience of the observer and the methods which he uses. Bearing in mind the limitation of the use of figures in this connexion, I have drawn the following conclusions from my investigations:

1. That at least 36.0 per cent. of the children from the poor districts of the City which I have examined were unhealthy or physically unfit, and as a result of this they appeared malnourished.

2. That since this high incidence of apparent malnutrition is not found in the children of better-class families, it is due to preventable causes.

3. In my opinion, the main *immediate* cause of the apparent malnutrition of the City children is the physical damage done by infective diseases occurring in young children at susceptible ages, and under conditions which prevent satisfactory recovery.

4. The main factors which promote and perpetuate this physical damage are probably:

- (a) The housing conditions which permit mass infections of young children at susceptible ages.
- (b) *Improper and inadequate diet, which prevents satisfactory recovery from their illnesses.*

It is probable that these two factors are of equal importance; but I would suggest that opinion on this matter should be reserved until a full inquiry, carried out by competent observers, in a scientific manner, has studied the problem more closely.



PART III

Children and Families

XI. *The Modern Decline of Breast-feeding*¹

PROSPERITY and social comfort have not encouraged maternal breast-feeding of the human infant. There can have been few women of fashion in the eighteenth century who did not hire a wet nurse to succour and suckle their babies. At that time the wet nurse was the mother-substitute, and failing a wet nurse the mother must feed her own infant or it would die. Now we have changed all that. Since then we have learnt how to feed babies on cow's milk in a simple and relatively safe manner. We have invented the feeding-bottle and the rubber teat. Chemists have shown us the composition of milk, and commerce has made it available for all. We can have it fresh, dried, or condensed. We know how much to give and how to give it in order to promote an infant's growth. So the cow is now the mother-substitute, and in this country there is no mother who cannot obtain this substitute if she so desires it. It is therefore an appropriate moment to ask ourselves to what extent there is a decline in maternal breast-feeding, and to inquire into the cause and effects of this decline. If it be found that a considerable proportion of women are gestating and bearing children but are cutting short this reproductive cycle by failing to lactate, it is surely both a matter of biological or clinical importance and a social experiment of great scientific interest.

The clinical importance of breast-feeding has always been recognized by paediatricians; but here again it is an appropriate moment to remind ourselves of this fact, for the infant mortality in this country remains twice as high as that in many other countries, and in some of our industrial towns it remains three times as high as it is, for example, in towns of similar size in Australia. Moreover, in some of our towns it has been slowly rising in recent years in spite of an increase in public expenditure on child welfare. This means that of the 40,000 deaths of infants each year in this country 20,000 might be prevented, or, to bring it nearer home, of the

¹ Read in opening a discussion in the Section of Diseases of Children at the Annual Meeting of the British Medical Association, Plymouth, 1938. Published in *British Medical Journal*, 2, 1938, p. 729.

social groups are breast-fed or artificially fed from birth. We desire to know also at what age weaning takes place. It is this precise information that is lacking. Some observers record their results by estimating the average duration of breast-feeding, others the proportion who are entirely breast-fed for 6 months or 9 months. But there is no commonly adopted system of collecting statistics which tells accurately when women wean their babies. From maternity hospitals we can obtain accurate information about the feeding of infants in the first few weeks of life. This in itself is valuable, for in this neo-natal stage proper nutrition and protection from inimical influences are more important than at any other age. Moreover, since a large proportion of babies are weaned shortly after this neo-natal stage the incidence of breast-feeding at this time tells a good deal about the trend of events.

In Newcastle Dr. Frank Stabler has made careful statistical studies of the clinical material in the Princess Mary Maternity Hospital in recent years. He has supplied me with the following figures, which represent the percentage of those infants leaving hospital at about the end of the second week of the puerperium who are wholly breast-fed:

1933	(1,431 infants)	87·8%
1934	(1,460 ")	84·3%
1935	(1,436 ")	80·9%
1936	(1,346 ")	74·6%

These figures from a city maternity hospital do not perhaps give a fair example of the people, so I have collected information from a private nursing home for middle-class patients and have excluded all patients with obstetrical difficulties or physically defective children. The matron is a sagacious woman with a strong personality who encourages breast-feeding whole-heartedly. Between 1934 and April 1938, there were 1,046 normal deliveries, and of these 76·8 per cent. of the infants left the home wholly breast-fed, 19·6 per cent. partially breast-fed, and 3·6 per cent. wholly bottle-fed. The majority of infants who are only partially breast-fed in hospitals are shortly weaned, so it is plainly to be seen that in this town satisfactory breast-feeding in the neo-natal period is maintained by only three-quarters of the mothers, and that there has been a significant decline in the incidence of breast-feeding in recent years. In Edinburgh Dr. T. Y. Finlay has made a careful

estimate of the incidence of breast-feeding by visits to the homes of three sample groups totalling 3,000 mothers, and has allowed me to use his results. The sample groups agree closely, and show that at the tenth to fourteenth day 87 per cent. of the mothers are wholly breast-feeding their infants.

There is an immediate decline in the incidence of breast-feeding when the mother resumes domestic and social duties. Dr. George Brewis has collected statistics of 1,326 infants who attended the municipal welfare centres in Newcastle in March 1938. Of these, 58.4 per cent. were wholly breast-fed at 3 months and 34.7 per cent. at 6 months. In Edinburgh, of the 3,000 mothers visited between the third and sixth months 55 per cent. were continuing to wholly breast-feed their infants and 38.5 per cent. when visited after the sixth month. In both towns the child welfare services give ardent encouragement to the mothers to breast-feed their infants, so the results may be regarded as on the highwater mark of achievement in big towns under prevailing conditions. A similar state of affairs obtains in other towns and other countries. In a south Lancashire centre in 1933 Wilson found that 50 per cent. of the infants were weaned at the third month. In an American village Bisal found that only 31 per cent. were breast-fed for 3 months. In Scandinavia two investigations have shown that 32 to 40 per cent. were breast-fed for 6 months. In Riga only 19 per cent. were wholly breast-fed for 6 months. In America conditions vary greatly. One observer writes: 'My conclusions show that breast-feeding exclusively for the newborn is about extinct in Brooklyn amongst the middle-class mothers.' Yet in Chicago, of 20,061 infants under the care of the welfare service from 1924 to 1929, 48.5 per cent. were entirely breast-fed for 9 months.

For our argument it is safe to assume that, apart from the little milk that may be yielded in the first few days, 20 to 30 per cent. of babies are artificially fed from birth in many if not most of our big towns, and that not more than a third of the mothers of these towns are fully feeding their babies until the sixth month. My experience is that bottle-feeding is a habit which readily spreads. If one woman in a maternity ward resorts to bottle-feeding others immediately begin to find difficulties in their own ability to breast-feed. So under prevailing conditions we can presume that a further decline in the incidence of breast-feeding will take place.

INFLUENCE OF FEEDING ON THE INFANT'S HEALTH

The medical officer of health whom I have quoted is not alone in questioning the superiority of breast-feeding over artificial feeding in maintaining the health of the infant, and the belief has spread to other quarters. Modern advertisements and other agencies have persuaded large numbers of women that artificial feeding is entirely safe and satisfactory for their infants and not disadvantageous to themselves. The mother-and-child subject favoured by the medieval painters has been replaced by the baby-and-bottle or the studio milkmaid subject of the modern photographic poster.

It is possible that hospital physicians seeing so much of sick infants are apt to exaggerate the dangers of artificial feeding. But, having taken that into account, it is beyond all question that breast-fed infants show a greater freedom from disease and a greater power of recovery from disease than artificially fed infants. The results in pyloric stenosis give straightforward evidence about this. In the Newcastle Babies' Hospital the mortality in 114 breast-fed infants with pyloric stenosis has been 5 per cent., but in 133 bottle-fed infants it has been 30 per cent. But the most complete evidence is to be obtained from the studies of Grulee and Sanford in Chicago. They investigated the morbidity rates and mortality rates in 20,000 infants. Including every minor disturbance of health, the morbidity rate in the breast-fed was 37 per cent., in the partially breast-fed group 53 per cent., and in the artificially fed group 63 per cent. The mortality rates were still more striking. Of their 9,749 breast-fed infants only 15 died, of 8,605 partially breast-fed 59 died, and of 1,707 artificially fed infants 144 died. These figures are significant enough to require no elaboration. But apart from the immediate significance of high morbidity rates and high mortality rates in artificially fed infants, there may be remote ill effects which have not yet been recognized or defined. It is possible that some of the serious degenerative diseases of adult life have their origin in the artificial feeding of infancy. I know of no investigation of this problem which has been scientifically carried out to disprove this, and we may recognize that such remote results are within the bounds of possibility when we recall the wonderful clinical observations of Nye, that temporary exposure of children to the paint dust on veranda floors of wooden shacks produced arterio-sclerosis and chronic nephritis 30 and 40 years afterwards.

BREAST-FEEDING AND THE MOTHER

There are difficulties in approaching this question of the effects of breast-feeding on the mother herself. Apart from the observation that repeated lactation in malnourished women may produce anaemia, there is very little scientific information on this subject. It might be suspected that women who have cut short the reproductive cycle by failing to lactate would be prone later to develop endocrine disorders. The pathological obesities which sometimes follow pregnancy suggest that a mismanagement of lactation may be an example of this. In the absence of scientific information opinions appear to be based largely on sentiment.

Much more is known about cows. Out of a common pedigree stock of shorthorn cows the breeders by selection have produced in 30 years two distinct strains—the dairy shorthorn and the beef shorthorn. Intensive breeding of these strains has resulted in a remarkable difference in their milking capacity. The dairy strain of shorthorn is capable of yielding 5 to 6 gallons of milk a day. The beef strain is capable of yielding less than 2 gallons of milk a day. So far has this gone that there is a danger that the beef-strain shorthorn will become incapable of yielding enough milk for her own calves. Selective breeding from human beings who hold the view that breast-feeding is no longer necessary may have remote biological effects we do not foresee.

The effects of lactation on the mother's character, temperament, and mental outlook deserve equal consideration with the physical or biological effects. This aspect has received very little study. Modern novelists, eager as they are to explain every conscious and subconscious gesture of their subjects, have hardly touched the theme. Havelock Ellis in his *Psychology of Sex* gives it relatively little notice. Yet the puerperal period is characterized by most interesting and distinctive psychological reactions. The desire for a mother to be left alone to sleep immediately after parturition has both a physical and a psychological value. And afterwards her peculiar jealousies and her craving for individual attention both suggest that she needs a special environment, for psychological as well as physical reasons. Again, if one is to judge by the look of lascivious content that comes over some women's faces as they feed their infants there must be a sense of achievement in the act which has a definite value in maintaining their mental health. These

mental states and emotional states of the lying-in woman deserve careful elucidation. Most of those whose profession it is to research or to write are debarred either by their sex or by their sterility from judging aright in this matter. But there are some who are equipped by experience and insight to undertake this work, and I would suggest that they should elucidate these problems for us. For my part I can only say that in most cases I can recognize a woman who has successfully breast-fed an infant, and that I find her to be a saner and a more sensible woman than her sister who has failed to do so.

THE PHYSIOLOGY OF LACTATION

If we are to discuss the causes of failure in breast-feeding it will be well to consider first the mechanism of lactation. New knowledge of the ovarian hormones and the pituitary hormones has filled many gaps in our understanding of this. During pregnancy the ovarian hormones develop and increase the breast tissue and the nipples, which in this way are prepared for lactation. Immediately after parturition the ovarian hormones which inhibit lactation recede, and a pituitary hormone, 'prolactin', then comes into play and establishes lactation. Later lactation is maintained by this pituitary hormone secreted under nervous reflex influences. There are at least two of these reflex influences. The first is the frequent emptying of the breast by suckling; the second is a nervous influence engendered in the emotional state of the mother and probably acting through the hypothalamus. Fear, doubt, anxiety, and lack of interest are states of mind which inhibit lactation. I have known a woman to be secreting daily 35 to 40 ounces of milk for her infant and yet the yield has fallen suddenly to 3 or 5 ounces a day under the influence of a dispute with the father of the infant. In my experience it is emotional disturbances in which the father is concerned that have the greatest inhibiting effect, but over-anxiety about her own ability to feed her infant is almost as harmful. The behaviour of the father during and shortly after the mother's parturition, although often a subject for ridicule, is significant enough to suggest a remnant of some remote biological purpose, and as such it has been studied by the anthropologists. For our purpose it is sufficient to say that in the early stages of

lactation he should remain an admiring and protective figure in the background, while his wife receives the ministrations and exhortations of a sagacious and experienced woman.

Studies of mothers' behaviour after parturition indicate that it alters in three distinct stages, each of them with a bearing on lactation. The first stage is one of profound rest and relaxation both for mother and child which may last for 24 to 48 hours. The child should not need milk or other fluid in that stage, and the mother should be free from meddlesome interference. It is one of the most miraculous of physiological arrangements that lactation is delayed in that early period and afterwards is suddenly established simultaneously with the awakening child's desire for food. The second stage is the establishment of lactation. This may take 5 or 6 days, and corresponds to the physiological lying-in period. In this stage the mother will require the help of the modern equivalent of the experienced and sagacious woman. Between these two the establishment of lactation should be a co-operative function, with the mother being allowed to fondle her child as often as she wishes, and without any fixed rules about 4-hourly or 3-hourly intervals. Another feature of this stage is that the mother requires to be segregated and free from distractions. She should, if possible, be in her own room, concerning herself only with the joys and duties of recovering from her labour and launching her infant on its new mode of existence. Studies of the peculiar jealousy reactions of lying-in women indicate the physiological need for this segregation. If in a surgical ward there are 12 women who have recently had operations for appendicitis we observe no jealousy and no unusual behaviour when a nurse gives particular attention to one of their number, but if there are 12 women in a maternity ward, all recently delivered, and a nurse has to give particular attention to one of them, there is immediately an atmosphere of jealousy which shows itself in demands for bed-pans and for other forms of individual attention. The third stage follows the lying-in period and begins when the mother, now out of bed, takes the feeding and care of her infant into her own hands. In these days its commencement, for many women, corresponds with the day of their discharge from a maternity hospital, abruptly to shoulder the double task of tending to their infants and their homes. Too often the significance of these stages and the need for the mother to pass successfully from one to another is overlooked. Particularly in maternity homes and

by maternity nurses are they overlooked. For 10 or 14 days the mother follows the ritual of a hospital bed-patient with her infant presented to her at the ordained intervals, and then on the next day full responsibility is suddenly transferred to her.

There are wide variations in the time and rate at which lactation is established. This is well recognized, but it is not sufficiently understood that lactation may have been prevented for several weeks and yet be re-established. I have seen it re-established 11 weeks after parturition in a woman who has resorted to bottle-feeding her infant, and who had not previously secreted any breast milk either by suckling or by manual expression.

There is a good deal of misunderstanding concerning the chemical composition of human milk. Fantastic theories of infant feeding have been built on the supposition that it is a food containing fixed and optimal quantities of protein, fat, and sugar. If this were fixed, there is considerable variation in its composition from one feed to another. The fat content of human milk is commonly 3 per cent. at the first feed of the day, 6 per cent. at the second feed, and between 4 and 5 per cent. at the later feeds. The protein and sugar contents vary also, but to a lesser degree. Even the volume of feeds fluctuates widely under natural conditions. A healthy baby may require and take 6 ounces at one feed and be satisfied with 3 ounces at the next. I wish to emphasize these facts, for I shall refer to them again when dealing with the causes of failure in breast-feeding.

Concerning the normal mechanism of lactation, there remains one other question. What percentage of women are capable of breast-feeding because of physical abnormality? Less than one cow in a thousand fails to lactate. Is the greater number of women who fail due to inherent structural faults or to environmental causes? In many rural districts in this country 95 per cent. of women successfully establish their lactation. There is no reason to think that with proper management the percentage of urban women capable of doing this is less than it is among the rural women. An endocrine mechanism which has enabled a woman to conceive and give birth does not at that stage fail to provide for lactation. There remain a few, less than 5 per cent., in whom it is physically impossible to establish lactation, either because of diseased or malformed nipples, or because the infant cannot suck on account of mental defect or of cleft palate or other physical fault. All women who are not handicapped by these physical

defects can breast-feed their infants if they desire to do so and if they are not prevented by an unsuitable environment.

THE FAILURE OF LACTATION

There are those who cannot, those who will not, and those who know not how to breast-feed their infants.

Those who cannot I have already mentioned. They are a small group with physical faults in themselves or in their infants. Those who will not breast-feed their infants I shall not discuss, except to say that often the fault lies not in themselves but in those who advise them. It is due as much to ignorance as to selfishness. It is fostered by a belief that bottle-feeding is adequate and breast-feeding no longer necessary. Modern methods of advertisement are *reponsible*, but the fault lies also in the readiness to receive advice and the desire which lurks in everyone to give advice about health and to appear possessed of occult secrets when giving it. The medical profession itself is not free from the influences of advertisement. No doctor receives a blotter once a week advertising breast milk. Too often do we hear of advice being given from within the profession that an infant must be weaned because the breast milk is not agreeing with it—advice portentously linked with a recommendation of a special brand of infant food, suggesting again the possession of a secret knowledge of the biochemical advantages of that brand of cow's milk over human milk for an infant who later may be found to be suffering from pyloric stenosis. More recently another kind of belief has arisen which encourages the weaning of babies. It is a belief that has grown out of the publicity about malnutrition and extra nourishment. It was illustrated for me by a young wife living on a completely adequate diet, who told me that she could not breast-feed her infant because she was not receiving 'extra nourishment' from a welfare centre. For this reason she had weaned her baby. The need for adequate nourishment of nursing mothers is obviously important, but here was a woman, typical of many in this country, muddle-headed by what she had read. In its effects this ignorance is the same as the selfishness of the young wife who will not feed her infant and explains it by saying that her husband wishes her to go about with him and does not like her to breast-feed—the wife who sacrifices the role of mother to that of mate.

It is those who know not how to breast-feed and fail in spite of their desire to do so who deserve our close consideration in this discussion. This failure ordinarily takes place chiefly in two stages. The first is in the lying-in period, through difficulties in establishing lactation. The second is shortly after the lying-in period, through difficulties in maintaining lactation.

I believe the chief cause of failure in establishing lactation is due to mismanagement. To explain this I can do no better than quote from a letter from Dr. Clifford Grulee: 'My personal opinion is that all this talk of the modern woman being unable to nurse her baby is due very largely to the modern nurse's tendency to poke a bottle of milk in the baby's mouth, which of course is due to the indifference of the physician.' He goes on to describe that in one of his hospitals they have two services for the newborn. In one, the out-patient service, 95 per cent. of the babies were being breast-fed at the end of the lying-in period. In the other, an in-patient department, less than 50 per cent. were breast-fed. They have been able to raise this to almost 90 per cent. simply by refusing to give the child a bottle in the first week of life. If fluid is required a little is given from time to time from a medicine-dropper. The attitude of the nurse at this stage is of supreme importance. I know of one maternity nurse who, after having been in practice for 15 years, came to a household in which she was first enthused about breast-feeding and then instructed in overcoming the difficulties of establishing it. Prior to that more than half her babies had been bottle-fed. When seen a few years afterwards she proudly proclaimed that since her sojourn in that house she had never failed to establish breast-feeding. The essence of the faults in most hospitals and nursing homes is that they attempt too much to mechanize breast-feeding. They have institutional rules about the times of feeds, the amount of feeds, the weighing of babies, and the test weighing of feeds to which they try to fit the patient. If this is not successful anxiety is aroused, and immediately the difficulty of establishing lactation is increased. I heard recently of a woman, troubled much by insomnia throughout her pregnancy, who was delivered of her child in the early hours of the morning and then fell into a satisfactory post-partum sleep, to be awakened at 7 o'clock to have her teeth brushed. The lying-in room is becoming too much a combination of a sick-room and a flower-decked reception-room. After lactation has started the mother should be

XII. *The Purpose of the Family*¹

THE STUDY OF THE FAMILY

IN accepting your invitation to discuss the duties you have undertaken, I must acknowledge that I cannot conceive responsibilities more onerous or more difficult than yours. They are the responsibilities of parents without the instinctive sanctions of parenthood, the burden of responsibility without the privilege of power. As all sensible parents are at times aware of their own shortcomings with their children, so must you in greater degree feel these faults, for you spend your soul and spirit without the sustenance of certainty. It is therefore a great privilege even to attempt to give you help in the task you are undertaking, for you represent that great body of men and women to whom is entrusted the near 5,000 score of homeless children in this country.

Throughout the ages the family in one form or another has been nature's chief device for fostering children. This philoprogenitive process of child care which lies latent or dormant in most of us, when awakened, seeks many outlets and takes many forms. Its object is to preserve life. However little we know about the purpose of life, it is at least certain that its preservation depends primarily upon a constant supply of young material which in health and by sensitivity, consciously and instinctively, can adapt itself to that purpose. If the quality of this human material falls too low, or if in later life it is turned aside too much from its prime biological function of progeniture to a pursuit of personal power, of adornment and of ostentation, then its civilization comes within the danger of decline and fall. The family is the eventual reservoir of this material and the place in which its qualities are preserved.

We can assume, therefore, that we should learn much by studying the family and by understanding its design and purpose. But it is not an easy subject to study, and to reach this understanding a conscious effort is required. A natural scientist, were he to pursue

¹ The Convocation Lecture of the National Children's Home, delivered on 8 July 1946. Published as *The Purpose of the Family: a Guide to the Care of Children*, London, Epworth Press, 1946.

this task, would take the structure of the family and consider it in all its forms. He would examine its variations. He would make a comparative study of family technique in different species and in people at successive stages of civilization. He would observe results and record aberrations and deviations from the normal. He might then use the methods of experimental science by observing arranged experiments, although that would be difficult in the human family. Nevertheless, he would have ample opportunity of observing these experiments which nature has arranged for him in a great variety of families, for no two families are alike. He would have ample opportunity also of observing adopted children, and children placed in nurseries, orphanages, and other institutions which society contrives as substitutes for the family. These would be his control experiments.

But most of us are not natural scientists, and we must use methods other than theirs to discover and know the principles of child care. Human endeavours pursued for a particular purpose lead to a practical art. Family life is thus one of the oldest practical arts in the world. What is the substance of a practical art? It is compounded of three elements. It is taught first in simple precepts and rules which are handed down; secondly in experience of its material; and thirdly by example. Wilfred Trotter says of this: 'The method of apprenticeship is thus the keynote of education in the practical arts, because it brings the pupil into familiar contact with his material, and gives him the constant example of his teachers in the actual things which he himself will ultimately have to do.' Apprenticeship in the practical art of family life is a lengthy process. It must be entered young, and is a generation's work. And the art degenerates when its simple precepts expand to learned treatises, or when theory replaces experience. Happily, theories and treatises are beyond my theme, which is to invite you to see what the structure and technique of family life may show, and to reinforce that when you can by experience in the company and constant example of capable craftsmen and craftswomen.

I must first attempt to clear the ground by mentioning some obstacles and difficulties which may distort our observations. It is in the highest degree desirable that we should approach our task as unprejudiced observers with dispassionate intellect and open mind. That would be the method both of the natural scientist and the eager young apprentice, but it is not possible without a moral

and emotional effort most of us cannot, or will not, make. And that is not surprising. Family and children are words carrying a high emotional charge, and we all possess within us a feeling mechanism which reacts to the mere mention of these words. It is notorious that feelings will colour an outlook, and these feelings will vary greatly according to our temperaments and to the experiences we have enjoyed or suffered in our own families. The extent of these reactions is to be seen in the universality of comment on a neighbour's or near relative's efforts to bring up her children. Nearly every man and woman is quick to indulge in this exercise. We must regard this as part of nature's intention to keep a concern for children simmering in each of us below the surface of our minds. If we recognize these difficulties in ourselves, and take them into account, we can pursue our observations with more profit.

There is surprisingly little written about the technique of family life, and about the adaptations which instinctively take place in parents and children to meet the changing scene. There is, of course, the literature of family life written in our great novels and biographies. But these are personal comments written by artists who, *ipso facto*, are men and women capable of deep feeling. How coloured these are by the authors' own experiences is plainly seen in books like those of Dickens and Charlotte Brontë. It is true that from Lytton Strachey onwards a new school of biographers has arisen which sets out to present a more objective view of family life. But these are no more free from coloured feelings than their predecessors. They differ mainly in being misanthropes instead of philanthropes. So you are left to draw your own conclusions from this literature of families, for it rarely descends to generalizations or to simple rules and precepts.

The social scientists have attempted to lay down rules of general validity. Anthropologists, sociologists, psychologists, pathologists, economists, and educationalists have all taken part in this. The anthropologists have used the family for their studies of primitive societies. From these they have developed hypothetical sequences with the Christian monogamous family as the end product and ideal type, and others placed according to the degree in which they differ from this type. The early evolutionists turned their attention to the family, and somewhat naïvely constructed a scheme according to which promiscuity was followed by group marriage, group marriage modified into polygamy, and monogamy was the final

step. The sociologists have looked at the family from another point of view, and used it as an example of a 'group' to argue their way to theories of social conduct. The psycho-pathologists, the economists, the educationalists, each of these has turned to the family from time to time, but in most cases they have been concerned mainly with end results in adult life, which they seek to explain by a study of children within a family, or seek to achieve by bringing early influence to bear on them.

There is little need for me to mention that politicians also have had their fingers in this pie, for we have recently had before us the example of a State perverting its families and children to its cruel purposes. But we must not be too squeamish or self-righteous. Every state throughout the ages has tended to exploit children and to justify its actions in political theories. Indeed, much that has been done in the name of children has been an attempt to prepare them early for a type of adult society conceived and desired on political or economic grounds. Nature changes slowly if at all. It is within the memory of man when industrial and political necessity demanded cheap body labour of children in mills and mines. We must therefore search our own hearts, and ask ourselves if we have changed so much for the better in so short a time. How much that is now done in families and schools in the name of education is but a preparation of children for mind labour in later life?

It would be churlish of me to leave the impression that the efforts of the social scientists have been in vain. I am concerned only to show that their line of country is not across our fields, that the study of the family requires a fresh approach by those trained in the method of the biological sciences, and that in the meantime the practical art of family life is best known through apprenticeship and experience.

Here I would make a parenthetical claim for two groups of people who are well provided with opportunities for the studies I have suggested. The first group is in danger of extinction. The second is struggling for survival. They are the village schoolmaster and the family physician. Both of these can dispose their lives to remain within a community for a generation or more, and so to know it well. They can combine their vocation with an intimacy of social life which is rarely given to those in other vocations. Armed with something less than the industry of a Gilbert White, or the patience of a Charles Darwin over his garden worms, they

should be able to give us the answer to many of the questions which face us. What are the needs of the family today? To what extent are extra-familial institutions such as schools, in pursuing their own aims of education, supporting or undermining the functions of the family? How much is the dissolution of family life due to the excessive individualism of the past half century by which men and women assert their right to live their own lives and to express their own personalities? Is not this excessive individualism the product of a mistaken conception of evolution? Does not human welfare depend on a recognition that the unit of human existence is not the isolated individual but the family?

THE SEVEN STAGES OF DEVELOPMENT

Let me first recall for you some simple facts about the development of children from one age to another. In some degree or other these should be the basis of common knowledge about child welfare and education. Alongside this I hope to show how parents develop those attitudes and sentiments by which the practical art of family life is ensured. We can then consider the ups-and-downs of family life, and from this serial story I hope some lessons useful to you in your work will emerge.

It will simplify my task if at times I differentiate between physical, emotional, and intellectual development, although this runs the risk of over-simplification because all three are closely inter-related, and it is difficult to define the limits of emotional activity. Some of the facts about development are simple, but the mechanism behind them is mysterious and wonderful beyond belief, or beyond anything the scientists have yet dreamed of. The concept I wish to give is that of a series of stages in a life ending at maturity, or ending at various levels short of full maturity. Another way to look at it is as a succession of seasons each complete in itself, but with its own needs for the growth, happiness, and education of the child in that season. In each of these stages or seasons there are particular dangers which may injure the children or the parents, or harm their relationship. It is best to keep the idea of maturity in the background. Do not regard the process as that of something small becoming gradually big. Let each stage or season be its own justification, complete in itself like the different larval existences of an insect. In this we may see, for example, that the stage of develop-

ment which rests between the ages of 7 and 12 is for many people the golden age of life, when we are most capable of living nearest to what nature expects of us. There was something of this idea in the adjuration to consider the lilies of the field how they grow.

The earliest part of life is automatic. It is the period within the mother's womb when from conception to birth the ovum grows nine hundred million fold. Judged by physical standards alone, the child before birth has already gone through by far the greatest part of its growth and development. Biologists divide this uterine life into a germinal period occupying about a week, an embryonic period of about 6 weeks, and a foetal period reaching to the end of pregnancy. I have called the development in these 9 months automatic, but we know that much physical harm may then be done to the child. To foster the child's welfare in that period the mother requires freedom from injury, freedom from illness, nourishment, and understanding. I stress the understanding, because towards the end of pregnancy a woman's outlook upon the world changes. She is in the grip of new emotional forces chemically engendered which are appropriate to the occasion. Forces she has not felt before, and will not feel until she is at childbirth again. At the time of birth, and shortly afterwards, the mother's physical and emotional mechanism is changing in subtle ways to meet the needs of her child. We require insight and experience to get this understanding, and, although it is outside my province to discuss it here, I must at least mention it for two reasons. There may be amongst you those who can influence public opinion to create maternity services which will take into account these emotional needs of women before, during, and after childbirth. The other reason is nearer to our subject. It is that a woman's capacity for motherhood may be injured by a misunderstanding of her needs at these times. It is in the early months after childbirth that a mother establishes a right relationship with her child. Or, putting it the other way round, before a woman can reach a confident relationship with older children and a capacity to deal with them successfully she must have experienced a sense of achievement and success in nursing and fostering babies in their earliest months of life. This is a point I shall return to later.

The different stages of a child's life after birth are more easily recognized, although since each stage merges gradually into the next any attempt to define them too exactly is artificial. The *first*

stage, lasting 2 to 3 weeks, we call the neo-natal period. In this stage the child needs care and nourishment, and the stimulus of close contact with the mother. The mother has equal needs, the chief of which are encouragement and the opportunity to fall physically in love with her child by close contact with it. This is helped by the presence of a wise and experienced woman, and it is all to the good when these qualities are combined in the person of the attendant nurse. But they are not created by the passing of examinations. In the background the father now begins to play a minor but important role. Instinctive feelings urge him to show pride in his child and sympathy with his wife. This attitude of the man at, and shortly after, the birth of his child is often a subject of *humorous comment*, but it has a *biological value*, and something is wrong with a man who through inhibitions or other faults cannot possess these feelings.

The *second stage* extends until the child is 6 or 7 months old. This again is a distinct period with its own special needs and processes, and the end of it is a transition to a different mode of living. The faculties of seeing, hearing, feeling, tasting, and smelling are then developed with miraculous speed, and in this each day is a new adventure. The raw materials for growth and experience must be amply supplied, and the environment must be secure. If these materials are lacking, or if the environment is harmful, the development may be checked, and the loss may never be made good, leaving the child less in stature and sensitivity than it should have been. The materials needed for emotional development are every bit as important as those for physical development. These are supplied by the individual attention of its mother, or of someone who acts as its mother. The father remains still in the background, providing only the sustenance of life. There are now reciprocal reactions between mother and child peculiarly adjusted to the occasion. In its waking moments the child demands companionship to provide that stimulus and encouragement which exercise its growing powers. The mother responds in a variety of instinctive ways to meet this need. She, and every woman like her, falls into the same habit of speech and noise-making with which she appropriately approves and disapproves, cheers and chides. She is emotionally altered for these purposes, just as much as a hen becomes another creature with the hatching of her chickens.

A biologist, looking on at this universal by-play for the benefit of

the child, would recognize it as a physiological process brought into action by internal secretions from the mother's endocrine glands. At this period she is absorbed in her responsibilities. Her sleep requirements are adjusted to the needs of the child, and her feelings are fully engaged in her new experience. How much this takes place has been revealed to me by a woman who, trained as a physiologist, has been able to record her feelings. She is a professional woman, rare in her capacity to combine a busy technical career with the management of a family and the pursuit of an active social life. She does this with eminent success which comes from a quick intelligence, a warm heart, and a power of rapid decision. She tells me that for a few months after the birth of a child, while she is engaged in its care and feeding, nearly every other interest leaves her. She is then unable to reach any decisions except those which concern the welfare of her child. She describes this state as irrational but deeply satisfying. She goes on to say that her capacity to judge the actions of men and women is reinforced by a sense of confidence and sense of achievement which these experiences with her children have given her.

I am dealing at some length with this attachment of mother and child at this second stage of development, because the welfare of children depends greatly upon it. The right relationship of mother and child at this stage is justified, therefore, not for sentimental reasons, but on biological grounds. I would not mention a matter so obvious to most of you were not the attitudes and sentiments of mothers in danger of being perverted by a cult of too much hygiene which makes them afraid, and by misreadings of Freudian theories which make them guilty. Under these influences many mothers deny themselves these natural outlets of feelings in contact and companionship with their children, and that leads to an atrophy of maternal functions and to worse.

I have had opportunities of studying these effects at close hand in the Babies' Hospital, Newcastle. Here, young children with critical illnesses were taken for treatment, and their mothers were admitted with them to nurse and care for them. Each mother and child lived together in their own room, arranged to give an atmosphere of domestic comfort which would not be alien to them. In the background there were experienced nurses and doctors to give technical aid when required. The mother was given as much responsibility as possible, receiving, for example, her child straight

from an operating theatre into her own arms. She lived with the child continually, and at all stages shared fully in the credit for its recovery. A contrasting group of mothers, with infants similarly ill, remained at home while their infants were treated in hospital. They endured the usual anxiety of separation and waiting which is common to those circumstances—a sensation of separation which, I suspect, is peculiar to the circumstances, and which no man and no woman who has not experienced it can appreciate. After a few weeks each mother returned to the hospital to receive her child at the hands of someone to whom she gave the credit for its care and cure. A study of these two groups of women over many years has revealed remarkable differences. In sharing the experience with their children, the first built up their confidence and enhanced their capacity. They returned to their homes and neighbours with a sense of achievement which stood them in good stead afterwards. The other group had a sense of failure which reacted unfavourably on the child. They were obsessed by the dangers of trifling symptoms. A necessary continuity of experience had been broken through being parted in those critical weeks of anxiety. I think it is possible to learn from this some simple lessons about responsibility, and about the pattern of social aids which parents need.

The age of 6 or 7 months is a transition from the second to the *third stage* of a child's life. This third stage lasts for about a year, until the child is near the age of 2. In one sense it is the hey-day of its life, when the child is living eagerly in an environment which is, or should be, almost completely devoted to it. It is a period of preparation before it is replaced by another child in the family, or passes out into a wider community. In this third stage of its life it demands instinctively those attentions by which it is educated, and receives them freely from all who come in contact with it. Discarding sentiment in this picture, we should examine it critically and appreciate its significance. Its purpose can be seen if we make the experiment of watching the behaviour of people admitted to the presence of a child 8 or 9 months old. Assume the child to be sitting playfully awake in an empty room. The person whose behaviour is to be observed enters the room. Immediately the child looks up to him, and he irresistibly approaches. He is now compelled to take notice of the child. He smiles, gestures, makes noises, and produces a bright object from his pocket or watch-

chain. Whatever may have been his mood when he entered the room, it is altered to the purpose of entertaining, encouraging, and interesting the child. If you now admit one by one a succession of people to the experiment, they will all behave similarly according to the mood of the child. If it is happy they will play with it; if unhappy, they will soothe it; if troublesome chide it. They will use the same language, make the same noises, and play the same tricks. These are not what the physiologists call conditioned reflexes. They are evidences of instinctive behaviour. A man who has never done these things before, or seen them done, will react in these ways. Persons who do not react in these ways are abnormal. The purpose is obvious. It is nature's compelling device for the education of children. If we accept the view that children of this age need this individual attention from one person and another, we must question the methods and results of those public nurseries where too many young children of the same age are kept together in a communal life which is imposed upon them before they are ready for it.

I shall describe four further stages after the age of 2, making seven in all. I shall do this in barest outline, mentioning only the chief features of each, so that they may be recognized in the later discussion of the design and purpose of family life.

The *fourth stage* starts with the child's first independent adventures before the age of 2, and ends with its first wilful acts of self-reliance at about the age of 4. It is an imitative stage. The child can be absorbed in its own play, and remain still unselfconscious. It now makes its first excursions from its mother's side, to watch the manifold activities around it. The child is now all things by fits and starts, but nothing very long. It has no sense of adult time, and should have none at this age. In spite of this commencing independence, the child needs the propinquity of its mother or someone else to whom it may go immediately for reassurance. The parent technique alters to meet new requirements, the mother playing one role, the father another. This is exemplified in the father's play, which now instinctively takes the form of hide-and-seek, and adjusts itself by alterations as the child gets older. The older children of the family or the neighbourhood enter into the child's life as the child enters into theirs, and older girls get here their first experience and responsibilities in child care in fostering children of these early stages.

The *fifth stage* lasts from about the age of 4 to about the age of 7. Habits are forming, personality is emerging. There is a new intellectual life vividly realized. Play is the essence of the period, and the child is not yet in the grip of the adult time machine. The *imagination is now awaking*, and *story-telling is one of the child's needs*, particularly at the end of the day, when his body is tired and he returns to the security of the parents' presence. This is a meagre but sufficient description by which to identify the fifth stage of a child's development. It is meagre because no adult can by recollection re-enter the mind of a child at this golden age of life.

The *sixth stage* lies in the years from 8 to 12, when boys begin their gangsterism in exploit, and when girls diverge from them in play. At this age they begin to create values, establish standards, and recognize ideal types. The outlines of character are formed, and, for good or evil, lines of conduct are laid down. Precept plays only a part in this. They get it also from a practical experience of human nature in relationship with their brothers, sisters, companions, parents, neighbours, and school-teachers. They see men and women at work unless they are absorbed too much at school. If they are lucky, they will see their fathers at work, but in modern life this happens too seldom. This sixth stage is a brief period before children are caught up in their self-made entanglements. It is a dress-rehearsal for life, in which they can engage in many physical and emotional activities *without injury to their feelings*.

The *seventh* and last stage of childhood covers the years of puberty after the age of 12 or 13, or a little later in some children. For the purpose of identification I need not describe it, because unlike the earlier stages of childhood it can be recollected by each one of us. We can remember what it feels like to be adolescent. The child then takes on the role of an individual personality, and assumes its responsibilities. It is a fumbling, furtive, and temporarily unattractive stage of life, when the apprenticeship is over, and before the skilled craftsman has emerged. The very unattractiveness of boys and girls at adolescence reveals the intricacy of nature's design and purpose. At that time it is a normal physiological event for spots to appear on the face, and for both sexes to be gawky and ungainly. In this design we may see the purpose of making the two sexes temporarily unattractive to each other, of holding them apart for a while until the grace and beauty of early manhood and womanhood bring them eventually together.

Each of these seven stages of childhood has its own rhythm of life and its own patterns. In these we see physical, emotional, intellectual, and moral development proceeding at different rates, alongside each other. There is an appropriate nourishment and exercise of body, feeling, mind, and spirit required at each stage. The essence of the art of child care consists in applying these in their proper order. Applied in the wrong order, forcing one type of development at the wrong time at the expense of the other, may produce effects that are the reverse of those desired. This may result in physical and emotional cripples, or intellectual and moral prigs.

THE FAMILY

In what follows I do not hark back to a sentimental approval of overcrowded families. Nor am I concerned with the falling birth-rate or with measures to reverse it, except in so far as families may become too small to fulfil the purposes for which they were designed. We must accept the established use of birth-control, and its implications in a society based on the monogamous marriage, but we cannot foresee the end of this sudden biological revolution in which the human race has involved itself. The immediate questions facing us are: can the purpose of the family be secure in spite of its restriction by the use of birth-control? To what extent can nurseries or other substitutes replace the family?

There is a tendency nowadays to regard the home merely as a training ground for citizenship, and the family as a community in which children learn their first social lessons. These are rather nebulous notions, and beg many questions about citizenship and communal living. I prefer a simpler outlook on the purposes of the family. It exists, first, to ensure growth and physical health; secondly, to give the right scope for emotional experience; thirdly, to preserve the art of motherhood; and, fourthly, to teach behaviour. Given these things, citizenship and community life will look after themselves. Without physical health and the environment that makes it, without emotional health and the happiness and security which go with it, without parental wisdom, and without capacity for neighbourly behaviour, there can be no citizenship or other social virtue.

A family at its best would have in it five or more children without

wide gaps in age between them. The parents at their best would come from similar families, but preferably not the firstborn nor the lastborn of these families. This variety gives the necessary range of characters. There are no star players on this stage, for all are equally stars. They play their parts and live their lives in a constant interchanging of relationships up and down the scale of the ages, and they do this within indefinable lines of conduct automatically accepted as their code. Although the acts are unrehearsed, the players do not go far astray, for they are under the influence of two unseen forces. One is an instinctive mechanism guiding the parents. The other is the culture of the family handed down in simple precepts, and rules from one generation to another, or less effectively by the example of one neighbouring family on another. The mother is the continuous thread throughout all this. Men create the materials and techniques for living. Mothers preserve the craft of living and transmit it from one generation to another. That is a woman's function as much as the preservation of the germ plasm within her ovaries.

The *first purpose* of the family is to foster the growth and physical health of its children. This is a concern deep-seated in the minds of parents, as anyone will know who sees welfare centres and doctors' consulting-rooms. In my experience this concern is felt equally by mothers and fathers, although it will show itself more in the mother, particularly with her youngest children. It is estimated that there are about 5 per cent. of parents, some of high intelligence, others of low intelligence, who are incapable of this concern. But the presence of these in our midst does not affect the argument. The mother is equipped for her duties by developing sensitivities to danger beyond the range of normal feelings. She will hear the whimper of a child in a distant bedroom when other ears are deaf to it. She will waken instantly alert to the needs of her child when strangers would do no more than stir slowly from their sleep. Mothers are guided also by rules about child health which in the past they have learned from older women. These rules need bringing up to date. It is one of the triumphs of our civilization that we have discovered enough almost to eliminate disease in childhood. We fail not through lack of knowledge, but through failure to spread knowledge and apply it. It is therefore the responsibility of parents to possess a simple knowledge about health and the nature of disease in childhood, and we can console ourselves

that this can be gained without engendering a morbid anxiety about ill health.

The principles of hygiene, the principles of nutrition, and the nature and symptoms of the common preventable diseases are within the comprehension of all intelligent people. The cause of a new baby forcefully vomiting, the danger of contact infection from a tuberculous adult, the criteria for clean milk, the symptoms of the common infective fevers, the simple methods of home nursing, these and a dozen other simple facts are part of the knowledge which mothers and foster-mothers should now possess. This knowledge is near at hand, and its facts are easy to memorize. We have advanced a considerable way in this health education, thanks mainly to the Child Welfare Services, to modern journalism, and to a few doctors who have interested themselves in this work. But this work has been uneven, and a great deal remains to be done. I hope that a future lecture in this series will be devoted to this subject of informing parents and foster-parents about the physical health of children and their common ailments and diseases. The principles of these will best be understood if we recognize that at each of the seven stages of development there are appropriate protections, appropriate nutritions, and appropriate exercises; that the first 6 months of life is particularly dangerous through contact with infection; and that much can be endured between the ages of 7 and 12 which would be harmful at any other age.

The *second purpose* of the family is to give scope for emotional experience. In the earliest stages of childhood this is gained entirely within the family. Later it extends beyond. Each of the seven stages has its appropriate experiences and adventures, and through these a child develops sensitivities, gains a working knowledge of human nature, and becomes educated in the art of living with other people. Thereby they create a capacity for companionship, for comradeship, for friendship, and for love. If a child's emotional experience is faulty at any stage of its development, it may to that extent be incapable of these things in later life.

The main difficulty in writing about emotions is in the inadequacy of definitions. When we attempt these we enter the field of terminological controversy. At one end of the scale emotions come near to reactive feelings, at the other end to acquired sentiments. Definition is made still more difficult by the alternative meanings of the very words we use. For example, pride in one

sense may be that wholly pleasurable emotion which a mother experiences on seeing her child take its first step, or watching her boy in his first race. In another sense it may be that state of mind which we recognize as intellectual arrogance.

Leaving aside all definitions, we know what we mean by joy, tenderness, hilarity, anger, rage, dread, suspicion, jealousy, grief, remorse, apprehension, fear, disgust, and so on. These are emotional states, each with its own physical sensation. We know that each of these states and sensations arises involuntarily at a physical or emotional experience, or at the recollection or imagination of these. We know that these experiences occur more often in relationships with people than with things. The particular physical sensations which accompany each emotional state are so peculiar to that state that we can presume a physiochemical mechanism within the body which operates each different emotion.

The view I wish to propound is that just as physical health must be developed at each stage by nourishment, play, and exercise appropriate to that stage, so it is with emotional health and emotional development. *This experience should not be directed continually to a cultivation of the pleasurable emotions.* It is a poor heart that never rejoiceth. Equally it is a poor heart that never weeps or melts in tenderness. Permanent happiness is a myth and, in seeking it as an end in itself, we may lose the means of finding it. It does a child no harm to experience the emotions of jealousy, dread, remorse, apprehension, fear, and sorrow, if only the parents are in the background to support and sustain. If the sorrow of death falls upon a family it should not be hidden from the children. *They should share in the weeping naturally and completely, and emerge from it enriched but unharmed.* But the physical presence and good example of parents or foster-parents are absolutely necessary, otherwise children will be painfully harmed and carry evidence of these injuries in their later years. In this lies the real apprenticeship to the craft of living.

The give and take of family life should provide this range of emotional experience without leaving any scars. In childhood we can bear to hear the truth about ourselves from our brothers and sisters *when we could not suffer it from anyone else.* By this very process children may be immunized to later dangers or fortified to meet them. Games, toys, and story-telling are the chief means to these ends, and the instinctive play patterns of young children

appear to be specially designed to exercise them in these ways. Take, for example, the readiness with which a 2-year-old child will enter into a capture and escape game with its father. It is indulging in the first sensation of apprehension followed by relief, emotions which are then entirely harmless for the very reason that the familiar father is the participator in the exercises.

In outlining a child's emotional experiences in its successive stages of development we can exclude the neo-natal stage of the first 2 or 3 weeks of life. To all intents and purposes the child is then still part of the mother, and its emotional life has not been awakened.

I have already dealt with the second stage of childhood at some length. It lasts until the child is 6 or 7 months old. In this period the infant should be the prime concern of the mother or foster-mother, and a binding relationship should then be established between them. To achieve this in full degree there should be no other infant of the same age to share this concern and relationship. This means that the child should not be living in a communal nursery at this stage. Its emotional capacity is now encouraged in manifold exercises by which the mother praises or chides, approves or disapproves, using gestures and sounds which are the universal language of all races. This relationship and encouragement are at their best if the mother is breast-feeding the child. This endows the mother with a confidence and certainty not to be gained in other ways. On these grounds alone, and apart from its benefit to the physical health, breast-feeding is justified. If the child must live without its mother, a substitute should be found to undertake the responsibility of individual attachment to each child in this second stage of its development.

In the third stage of childhood, lasting until the age of 2, the child should still be the only pebble of its kind on the family beach, claiming and receiving the constant encouragement and admiration of its parents. In a natural set of circumstances it would *shortly be displaced by the birth of another child*, but for this brief period it remains the centre of care and attention. The child is still unselfconscious, so no harm will come from these devotions. By cuddling and fondling, by a little teasing and much coaxing, its capacity for feeling is educated. The simple reactions of joy, fear, apprehension, and relief are now established. But these experiences are short-lived. They are trial flights in emotional experience which

end safely if the reassurance of the mother's presence is near at hand. Fathers and older children play an important part at this stage, for it is through them that the child emerges out of the world centred on its mother into a wider field of human relationships. It is a period of venturing forth in cautious steps.

In the fourth stage, between the years of 2 and 4 or thereabouts, the child's emotional experience widens. It passes then into the self-conscious stage, and becomes capable of shyness. It participates in the communal life of the family without entering fully into it. It now needs the company and example of older children. This is the debutant stage of childhood watching all that goes on around with wide-open eyes. But the child still responds to cuddling and fondling, and will find a way to seek from its parents its individual share of these at odd moments of the day.

Some of the child's emotional reactions are now fixed to experience. The fear of a stranger, the joy of a father's return, the anger of deprivation, the happiness of discovery—these are the very substance of a child's life at this stage. If these are to be assimilated and turned to good account, the child requires the propinquity of its mother, or of someone else in whom it can immediately find reassurance if the need arises. At this age a child interprets its new experience by reference to her or to some other trusted person. If a bomb falls, the child turns not to the scene of the noise, but to the expression on its mother's face to seek interpretation of the event. In the same instinctive way a mother in the presence of danger turns to her youngest child regardless of herself and all else. This interpretative function is one of a mother's greatest contributions to a child's welfare and emotional education.

Play, which is an all-important part of a child's emotional development, now takes two forms. These are the beginnings of group play with three or four other children, and the solitary play which finds outlet in odd activities. There should be no disciplined regularity of these forms of play, because the child is not ready for marching and parading even if these be arranged to the allurements of a flute, or to the tinkling of a piano. Before the age of 5 the confinements of time, space, and organized communal routine must be avoided as much as possible. Living within the protectorate of the family, or in a nursery arranged as near to that pattern as possible, the child should be free to enjoy its own solitudes and its own engagements with a variety of characters from the milkman

to the street musician, from the grandfather to the baby sister. At this period, and for the last time in its life, the child is completely uninhibited in entering into these relationships. This opportunity must not be withheld from children, and it is for this reason we must question the wisdom of absorbing too much of their days in nurseries and nursery schools where variety is lacking. Village life, with its opportunities for standing and staring, provides this rich variety unhampered by time schedules. The problem in urban life is to provide these same advantages and opportunities.

The fifth and sixth stages of childhood carry a child from the age of 5 to the age of 12. It is the golden age of life before puberty brings its new significances. Every day should be a new adventure, unclouded by a brooding future and free from a worful past. The child is now conscious of its own personality, and is questing in every direction for ideal types to imitate. That so few children enter fully into the warmth of this golden age is one of the tragedies of our way of living. The fault lies mainly in the ambitions of so many parents who have designs on their children's careers, and of those schools which lend themselves to the same purpose.

I am not pleading here for discipline-free schools and homes. Children between 5 and 12 welcome discipline if it is just and axiomatic. It simplifies their actions and relieves them of responsibilities which they are not ready to bear. But within this discipline there must be freedom and versatility. Play is the essential activity, and year by year this becomes adjusted to new obligations, responsibilities, and intellectual interests. It should be organized as little as possible by the children themselves, as will happen naturally if the opportunity and time be provided.

In the companionship and competition of these older children find much of their emotional experience. In this way they can become familiar with the sensations of love, happiness, remorse and joy, anxiety and relief, tenderness and hostility, fear and security, pride and shame. These are experienced at this age in a manner which gives an opportunity to enter into human nature without doing injury. The family, and the community outside the family form the best arena in which these reciprocal parts are played out. As the children are chosen, play reciprocal parts in the family form. The parents have a skill in these arrangements which is a true genius. They are usually people who do not share...

into their children's daily lives, but impose just rules of conduct and demand commendable standards of behaviour. They are nearly always tolerant and unselfish people, achieving their results more by example than by precept. Their relationship as husband and wife provides the most important of these examples in its display of courtesy and chivalry. Against this background their children's emotional lives will pursue an even course, however violent their fluctuations from hour to hour.

The natural grouping and regrouping of these older children in their different forms of play is biologically interesting, and has all the appearances of design and purpose. The boys depart to arrange their own games and gangster play, and the girls on their part pursue their own bents. Here again I would draw attention to the natural tendency of older girls to seek out a child of 2 or 3 whom they may foster. In the same way a boy will sometimes seek out his younger brother to become his guide and philosopher and friend. I emphasize this because I believe that the group in each of your cottages and homes should be arranged to imitate the family in this respect, and include the children of different ages in order to provide the oldest of them with this opportunity of developing their capacity for tenderness and care.

The seventh stage of childhood carries the child into the new emotional world of puberty. Up to this point children may have been active, eager, and friendly, now they become reticent and quiet. They require more sleep at that stage than at the preceding stage. Their interests change. A girl who has been devoted to play, and has sought every opportunity of giving help to her mother, now goes cold on these duties. For this short period she is not domestically minded. She is busy with her own emotional interests. Applying the principles that each stage of childhood must have its own appropriate experiences, and that an experience inappropriately applied may produce effects which are the reverse of those desired, parents and teachers should understand the particular needs of boys and girls at this age. For example, it appears to me to be wrong to teach domestic science and 'mothercraft' to girls of 14. They are ready to receive that instruction before that age, and will return to it again later, but at puberty they are chiefly concerned about themselves, and it would be preferable to teach them the art of dressing and deportment at that age.

I shall here summarize this description of emotional life in the

different stages of childhood. In each of these a child should have its full experience, from cuddling and fondling in early infancy to happy competition and serious endeavour in later childhood. It is part of the parents' duty to arrange and control family life to these ends. This should be done unobtrusively. The children must adhere to a few daily routines, but otherwise they should have time, opportunity, and freedom to plan their own activities. In this way a wide range of emotional experiences can be assimilated and turned to good account in the successive stages of childhood, but only if they are gathered against a background of parental love, tolerance, and example. Side by side with this there should be a code of moral conduct authoritatively applied in early childhood, and reasonably accepted in later childhood. Here again the essence of success is to apply this code in a manner which is consonant with the age and nature of each child. In the earliest stages mother and child should be bound in a close physical relationship. That is the foundation of emotional education. Stage by stage new experiences are added by contacts with the father, with brothers and sisters, with neighbouring children, and with various adult types of men and women. The family exists to exercise children in these experiences, and to provide a place in which they may argue and talk about them before the reticence of puberty falls. The parents exist to sanction and to interpret these experiences. The aim of emotional education and experience is to reach a maturity which renders us capable of enjoying, enduring, and understanding human relationships in later life. The immediate purpose is to nourish the child, and to supply him with a preliminary knowledge of the raw material of human nature. There can be no wisdom in later life without this knowledge.

THE ART OF MOTHERHOOD

If there were no mothers there would be no families. It is equally true that if there were no families there would be no art of motherhood. It is the *third purpose* of the family to preserve this art.

A woman does not become a mother merely by giving birth to children. Motherhood in the sense in which I use the word includes the art of encouraging and controlling children. It is a practical art. It may be highly developed in a woman who has no children of her own, but it is seen at its best when a mother reaches her maturity

with an experience of six or seven children behind her. From each of these she has learned something. If you watch a mother in the fullness of this maturity, you will see her dealing with her children with certainty and instinctive quickness. For each child according to his age and mood she uses the right touch, the right attitude, and the right word. She will turn to her petulant schoolboy with a little chiding, to her puling infant with tender fondling, to her distressed schoolgirl with ready praise, to her 5-year-old child with patient encouragement. She will use reproof, praise, sympathy, laughter, and all the other instruments of emotional control and guidance. *In the midst of her daily life she conducts this orchestra of many players in many moods.* There is little need for me to say that to be completely successful in this responsible task she needs a husband who will courteously and chivalrously provide both shelter and protection, and sustenance for her mind and spirit.

Some may regard this as a romantic picture of motherhood, but I will counter this by saying that it is attained in many families at all levels of culture. That so many parents fail to attain it, is in some part due to a failure to present this picture clearly to them as an *ideal capable of realization.* The tendency nowadays is to exaggerate the economic difficulties of motherhood, to depict its tribulations, and to belittle its compensations and rewards. Much that passes for social aid to mothers is construed in a way which raises their fears and undermines their confidence. They are relieved of their children when they should be relieved of their chores. They are tempted to wage-earning when they should be paid to make a home. The core of the trouble is that our economic system is not based on a philosophy of human welfare which recognizes the right of every mother of a family to possess the means of home-making if she so desires it. To use Sir William Beveridge's words, '*A family still remains the greatest single cause of poverty*', and it will continue to be so in spite of the family allowance which is shortly to be given. The trend of our political and social life still tempts parents to prefer a motor-car instead of a child, and this will not be altered by dangling before their eyes the bogey of a declining population.

The receding age of marriage is another factor which undermines motherhood. This also is economic in origin. It has two effects. Firstly, it postpones the age of child-bearing beyond the period of greatest fertility, and, more important still, beyond the

age of woman's greatest sensitivity to the lessons of motherhood. Secondly, it increases the proportion of spinsters who seek outlets in professional tasks many of which are substitutes for a mother's work. If mothers were provided with greater facilities and more ample means for home-making, we would to that extent need fewer women in our social services. In spite of these handicaps motherhood remains the most rewarding occupation in the world. Children who are reared healthy, sensitive, and courageous are indeed hostages to fortune.

Much of what I am saying on this subject of motherhood has been said so often before that it has become commonplace, but I wish to stress one technical aspect of it which may be important to some of you concerned in providing institutions for homeless children. I have said that a mother requires the help of a husband who will courteously and chivalrously provide shelter, protection, and encouragement. I believe that this relationship has a profound biological significance. It cherishes a woman and inspires her in her endeavours. If this principle be applied to institutions for homeless children, it would lead to the appointment of men advisers or supervisors to provide the same kind of support and encouragement to the foster-mothers who are in immediate charge of children. Suitable men may be difficult to find. They should be chosen for their sincerity and courtesy, rather than for their administrative ability. Most suitable men will not readily take on these duties as a whole-time professional occupation, but a partial substitute may be found in a medical practitioner who will be near at hand to undertake the responsibility of advising and helping a foster-mother in the same manner as a family doctor can help the mother of a family.

FAMILY BEHAVIOUR

The *fourth purpose* of the family is to teach behaviour. I have already touched on this in saying that children should gather their experience within a code of moral conduct authoritatively applied in early childhood, and reasonably accepted in later childhood. But it goes further than the children. The parents themselves are altered for better or for worse by the manner in which they play their parts within the family.

Some may argue that I am putting the cart before the horse.

They will say that a man's character is fixed before the age of marriage, and that as he was before he became a father so he will be afterwards. A bad man will become a bad father, a good man a good father. In the same way they will argue that a selfish girl will become a selfish mother, a lazy girl a lazy mother, and so on. But this is only half the truth. The outlines of character and behaviour are, of course, laid down before the age of marriage, but for many years afterwards the care of children engenders qualities which have a permanent effect on the character and behaviour of men and women. A woman may never learn true patience and tolerance until she has exercised herself in these things through a responsible care of children. A man may remain selfish and arrogant until these faults are softened by his responsibilities as a father. Chivalry and courtesy, qualities I set great store on, will reach their greatest heights in a man who exercises them constantly in his own family. The effect of this discipline on character and behaviour is seen in the example of the sympathy and unselfishness aroused by the sight of a lost or homeless child. If this happens in a community of neighbours by the death or desertion of parents, the woman who will most readily come forward to give a home to the lost child is one who already has a large number of children. It is no uncommon thing for a mother who has ten children to come immediately forward to undertake this responsibility, when the woman with a single child will stand aside. This capacity for philoprogenitive feeling grows on what it feeds on. The more it is exercised the greater it becomes. Shakespeare went far in saying, 'Man's nature is subdued to what it works in', but we cannot doubt the truth of this if we observe the mellowing influence which the responsibility of children has on the nature of men and women. To watch the manner in which they fulfil their parental function is in the end one of the finest tests of maturity and character. It is a test also of a man's capacity to manage his affairs outside the family.

When we speak of behaviour within a family it is, however, the parents we usually have in mind. In studying the behaviour of the individual I would suggest that we regard it in the same light as we regard his physical and emotional health. It is a condition to be cultivated by stage by appropriate nourishments and exercises. Just as the physician gives nourishments and exercises for physical health, others for mental health, so moral health has its own needs. Here the same important principle applies that what is good for one

stage of childhood may be harmful for another. It is not a case of getting a child as young as possible and fixing its character early. It is not necessarily true that as the twig is bent so will it grow. If bent too early it may break. If bent too hastily it may splinter. If bent too late it may spring back.

Each stage has its appropriate needs, and the art of teaching or developing behaviour in children is to know when and what to do. If Huxley is right in saying that 'Good is a matter of moral craftsmanship' then that parent is a successful craftsman who knows that to force communal behaviour on a child before he is ready for it may produce unexpected and undesirable results in later life; to enforce too early a moral code of truthfulness may be as harmful as neglecting it until it is too late; to demand unselfish sharing of toys at 2 years old may be as harmful as excusing it at 10 years old.

If we knew as much about behaviour and moral health as we do about physical health, we would take the seven stages of childhood and describe what could be done in each of these to encourage development. We would measure moral growth. We would become familiar with the childish moral ailments and know which were transient and which were dangerous, which were curable and which were incurable. With experience we would be able to recognize which moral illnesses would confer immunity from later trouble, which would spread like infections, and which leave permanent scars.

I am not speaking lightly when I use physical health as the analogy of emotional and behaviour health. I believe that the same principles will hold good in all these fields. A few children will become physical cripples because of inborn faults, others because of illnesses and accidents imposed upon them before they are resistant or immune; but apart from these occasional accidents the body conforms to a law of natural self-cure if it has been prepared and nourished rightly. It is the same with moral illnesses and ailments. Children must be protected from some moral illnesses and nursed through others by individual care. That way lies immunity and health. Were we masters of these rules we could reduce behaviour to the level of a moral science. But we are far from that, and will not reach it until we have found a modern Vesalius and a Harvey who will describe more clearly to us the anatomy of behaviour in its different stages of development, and a modern Sydenham who will describe more clearly for us the nature and

source of moral illnesses and behaviour faults. *Psychology* has not taken us that far yet. It is still in a pre-Vesalian state.

It strikes me that the Church might undertake this task, and their Sunday schools might then become places of enlightened study of these problems. They might study, for example, persistent untruthfulness, and determine its effects in later life, in the same way as a medical scientist might watch the progress of a chronic illness. They might find out for us at which stage of development particular disciplines are best applied. They might find that moral and intellectual priggishness are caused by disciplines inappropriately applied at the wrong stages. By observation and experiment they might discover at what stage of development a child is capable of appreciating an ideal type of behaviour, and how that should be represented to them. In doing this, I feel sure they would discover the need to lay the same emphasis on a type of ideal womanhood, as on a type of ideal manhood, for it appears to me to be a fault of most religions that they present only the ideal man for imitation, and too often they do it in a manner which appears irrelevant to women and to children.

Until these moral teachers come along and help us in a more precise way, we must rely on the family itself for training and for teaching behaviour, with the school playing a somewhat remote but important part in the background. Here we must fall back on the methods of the practical arts which I mentioned earlier in this lecture, in which apprenticeship is the keynote of education, with the children exercising themselves as brothers, sisters, and neighbours in the behaviours they will have to follow in later life, under the constant example of the master craftsmen who are their fathers and mothers. As in all practical arts, they will be helped by simple precepts and rules, which must be sorted out from the multitude of sayings of every moralist from Confucius to Kipling.

As one whose habit it has been to study the nature of some of the diseases which man brings upon himself, I would only say that we fail most often as examples to our children through parental indulgence, parental indifference, and parental selfishness. But this needs careful interpretation, for it all depends on the stage of development which our child has reached. Indulgence may be a virtue at one stage and a vice at another. Indifference and selfishness are never justified. They are the cold winds of parental behaviour which may blight a child. But something near to in-

difference occasionally has its place, and this was no doubt meant by a now outmoded moralist who said, 'It is the business of parents *mentally* to forget but dynamically never to forsake their children, an active and strenuous business which must not be shelved off to a stranger.'

I will say no more about family behaviour and parental example, but end with another's words:

We of this self-conscious, incredulous generation seek to sentimentalize our children, analyse our children, think we are endowed with special capacity to sympathize and identify ourselves with children. And the result is that we are not more childish, but our children are less childlike. Know you what it is to be a child? It is something very different from the men of today. It is to believe in love, to believe in loveliness, to believe in belief.

So said Francis Thompson, remarkably for a childless man, but he, like many of you, had great experience of family life in the places where he had lived.

THE FUTURE OF THE FAMILY

So far, in broad strokes, I have attempted to depict the family as a place of warm activity which fosters the welfare of children and the wisdom of parents, a place in which an apprentice experience of life is gained, a home with a tradition, a school of character and moral concepts, a device well suited to its purpose through countless ages of trial and error, of successes and failures. Now, and perhaps for the time being only, we are saying good-bye to all that. Families will be restricted in size. In the next generation the majority of parents will themselves be the products of these restricted families. Their childhood apprenticeship will differ from that of their ancestors. This denotes a mutation of prime biological importance. It is worth while examining carefully its causes and effects.

The primary causes are obvious. Families will decline through the use of birth-control, and through the postponement of marriage beyond the age of a woman's greatest fertility. The subsidiary causes are the status of marriage altering to that of a companionate association, the parents' desire for a greater freedom to live their own individual lives, and their desire to have fewer children in

order to spend more on the education and equipment of each child. All these causes do not operate universally. Already, I notice that more young parents of the professional class are deciding to have families of five or six children than was the case 10 years ago. They are doing this in spite of the economic uncertainty of the future, and in a belief that in that way lies greatest happiness. They are regarding their marriages as a social contract, and not as an erotic episode. But these are straws in the wind, and do not yet indicate a general reversal of opinion or policy. They will form a small aristocracy of great social value, and it will take a few decades for their example to act on others.

We must therefore face the decline of the family. The effects of this will be standard families of 2 or 3 children, the failure of the family to provide the full experiences for the development of its children, and a search for social instruments outside the family to substitute these experiences. The ultimate effects will depend on man's capacity to adjust himself to these changes with wisdom, intelligence, and faith.

All these changes are not necessarily disadvantageous. We shall reap a little reward in a lower infant mortality, and an improvement in the physical health of children. Education may ultimately lead parents and educators to consider in what the true welfare of children consists. Schools may follow their present trend of widening their conception of education. Beyond that we cannot see. But it is within the bounds of possibility that our civilization will decline through a failure to preserve and promote family life and the neighbouring society in which it should be set.

The crux of the matter is the attitude of the State. Up to the present the State has always been a supporter of family rights, and in its encroachments on parental authority, or upon family feeling, it has simply obeyed an irresistible necessity. But up to the present, legislators and government officials have been brought up in families which have been large and humane. From now onwards the proportion of bachelors, spinsters, and only children in our governing class will become larger and larger. Most of these will be people who have been trained mainly in politics and in economics. Few, if any of them, will have studied biology, which is a necessary component in the intellectual equipment of people who are legislating for the welfare of children. We must therefore remain apprehensive if power rests in the hands of people inexperienced

in family life whose political philosophy is based on economic theory. They may not understand whether newly created social instruments such as nurseries, nursery schools, and community centres will benefit or harm the family.

More than fifty years ago Charles Pearson wrote a book on national life and character in which he predicted

a state of things in which marriage will be contracted without reflection and broken up without scruple, in which children will be cared for when they are young with even more tenderness than of old, but with incomparably less anxiety to fit them for the moral obligations of life, and in which the claim of parents to be obeyed will cease with the children's need of support. Family life will be a gracious and decorative incident in the system of such a society, but the family as a constituent part of the State, as the matrix in which character is moulded, will lose its importance as the Clan and City have done.

To a biologist 50 years is a very short space of time, and we cannot yet see the end of the journey we are taking. Plato's conception of a commonwealth in which children are to be taken from their mothers and brought up by the State, and the example of Rousseau's experiments with his own children, fell on stony ground. The instinct of parental love is so intimately associated with our nature that we cannot imagine it will ever die out. But we must recognize that the modern State may be governed and administered by men and women whose parental instincts have atrophied, and who have little or no experience of family life.

At all stages of civilization the family has merged itself in its neighbouring society. It has been part of the tribe, the clan, the manorial village, or the country town. This association of family with neighbouring family produced its own forms of mutual aid and social security. Until the nineteenth century the State did not encroach upon the family, and limited its actions to the care and control of vagrants and delinquents. Beyond that there was no social legislation.

The nineteenth century brought three revolutions which changed life. These were the conception of equality and liberty which loosened the social structure, the idea of evolutionary human progress which loosened religious beliefs, and the rapid growth of industrial towns which dissolved neighbourly relationships. Mankind then struck its tents, and has been marching hither and thither ever since. The change which most concerns us here is the

dissolution of neighbourly relationships. Year by year an increasing proportion of our families come to live in towns alongside other families with which they have no intimacy, and for which they feel no obligation of mutual aid. This change for the worse is hastened in many places by the flight of those citizens capable of social leadership from the towns in which they work to the dormitories in which they sleep. The tragedy of the Jarrows between the wars was not their unemployment, but the absence from those towns of people with wit enough to make unemployment endurable, and means enough to create their own mutual aid.

Man is a resourceful animal, so it is not surprising that he reacted to these alarms and excursions of the nineteenth century in a variety of ways. For the welfare of homeless children your great voluntary societies were created, and the State came forward with new social legislation. Schools, orphanages, hospitals, welfare clinics, nurseries, school meals, kindergartens, and nursery schools were amongst the instruments provided. Late in the day the much belated reform of family allowances was made. Most of these instruments were hastily devised, and many quickly outlived the occasion of their need. The time has now come to review and reshape them. Accepting the premiss that the purpose of the family must be secured, we must ask ourselves to what extent they are aiding or hindering the family and the spontaneous neighbourliness in which it should be set.

I cannot here enter into a discussion of these various social aids to the welfare of families, but will conclude with a personal affirmation. Anglo-Saxon civilization has been built out of the character and faith of its people. In the past these qualities have been engendered by the influence of family life on children and on parents, and developed by their experiences in companionships with other children and other parents. We of this generation through the decline of the family are witnessing the most sudden biological change the human race has known. The dangers of this sudden change are intensified by the decline of neighbourliness, by the encroachments of State institutions on the family, and by the spread of a materialistic individualism. The complexity of modern society commits us to an increasing arrangement of our lives by the State. Anglo-Saxon civilization will decline unless people re-create natural neighbourliness, and unless the State bases its actions on a philosophy of human welfare which recognizes that the unit of

society is not the isolated individual but the family. To that end the purpose of the family must be known and realized, and our social legislation, our methods of education, our arrangement of mutual aid, and our spending of public money must be adjusted to that purpose.

XIII. Family Studies in Preventive Paediatrics¹

WHEN John Clarence Cutter founded this lectureship and decreed that the lecture must be delivered in Boston and free to the medical profession, to the public, and to the press, he was probably prompted by the zeal for education that had taken him many years before to his teaching post in Japan. But as I look back I believe that he was influenced also by Lemuel Shattuck's *Census of Boston*, a book so famous that a British historian has recently described it as 'one of the most remarkable documents in the annals of public health, which might serve as an ideal even today'. While I handled that book in your library this afternoon, as a runner might make a trial sprint before his race, I could only hope that, in attempting to describe some field studies in medicine and their application to clinical education, I might catch a little of the spirit of that book and these two men.

In this Cutter Lecture, with its honourable history of discovery so well expounded year by year since 1912, I need say little of the history of preventive medicine. Nor need I dwell for long on the great achievements of paediatrics within that field of human endeavour. We have travelled far from the eighteenth century, when Gibbon, himself the sole survivor of seven children, could write of the death of an infant as 'an unfortunate but highly probable event'; far also from last century, when a third of the children died before they reached the age of 10; and even from the beginning of this century, when nearly a fifth of the children born in our towns died within a year. The story from that point is a record of advance and victory until now the death of a child is regarded as a calamitous event that must be inquired into. The major pestilences are under control. Our infantile mortality in good environments is nearing an irreducible minimum. If there is still a pitiable waste of life in the neonatal period, the health of older children is remarkably secure. Taking neonatal deaths at a level of 20 per 1,000 as a basis, there are *pro rata* more deaths in the first month of life than

¹ The Cutter Lecture on Preventive Medicine, delivered at the Harvard School of Public Health on 21 May 1949. Published in *New England Journal of Medicine*, 243, 1950, p. 205.

in the whole next 20 years of life. In England, with its 42 million population in 1945, only 899 children died at the age of 5, and 438 at the age of 11. There is only a 1·19 in 1,000 chance in any year between 10 and 15. In the twelfth year of life, when death-rates are lowest, the chance of dying is 1 in 1,280. This may be too optimistic a picture, and I do not wish to suggest that there is not more yet to be done. We are left with preventable deaths and disablement in childhood that come from trauma, from genetic faults, from tuberculosis, and from the uncontrollable infectious diseases that remain with us. We are faced also with the dangers that will arise in our attempts to adjust ourselves to our new technologic and comfort-providing environments, as is seen, for example, in the new epidemic constitution of poliomyelitis.

Against this achievement of preventive paediatrics in controlling disease and fostering health we must set the diminishing birth-rate. In any final act of biologic accountancy this must be taken into consideration, particularly in judging the effect of the diminishing birth-rate on the size of the family and the subsequent effect of the small family on the virtue, character, and mental health of parents and children.

Many hands have gone to make this progress in preventive paediatrics. But as a clearer understanding of a problem is best approached by a clear analysis of the facts, I should pick out first for praise the inquirers who have used statistics to reveal the facts. The story of these begins with Graunt, who first published his *Natural and Political Observations on the Bills of Mortality* in 1662. He had to work with crude machinery, which he describes as follows in his sixth edition of 1676:

We have hitherto described the several steps whereby the Bills of Mortality are come to their present state. We come next to show how they are made and composed, which is in this manner, viz: When anyone dies, then either by tolling or ringing a bell, or by speaking for a grave of the sexton, the same is known to the searchers corresponding with the same sexton. The Searchers hereupon (who are antient matrons sworn to their office) repair to the place where the dead corps lie and by view of the same and by other enquiries, they examine by which disease the corps died. Hereupon they make their report to the parish clerk, and he every Tuesday night carries in an account of all the burials and christenings that week to the clerk of the hall. On Wednesday the general account is made up and printed, and on a

and from one generation to another. This was the method of Jenner as it is the method of my neighbour Dr. Pickles, a predecessor of mine in these lectures. But not many men in general practice have the time or training or inclination for such work. In paediatrics we shall need this method if we are to study the genetic constitution of disease, and the epidemiology of some of the indefinable infectious diseases of infancy. It will be a necessary method also when we come to study the techniques of family life, the capacity of parents to play their part in preventive paediatrics. This thought must have been in the mind of the writer of a plaintive letter in the *British Medical Journal*, who said, 'An intelligent community that really wished to improve its health would put its best doctors into general practice and give them sufficient leisure to research into the problems of real life.'

But the isolated inquiries of a medical practitioner are applicable only to limited subjects. More extensive research may be necessary for other particular purposes, and for these a more complex organization will be required. This brings me to speak of a personal experience of field inquiries in clinical preventive medicine designed to be a component part of the work of a university department of paediatrics with a comment on its effect on the teaching and practice of paediatrics.

The Medical School and Teaching Hospital of my university is in an industrial city of 300,000 people with a contiguous and neighbouring population of over 1 million. The Paediatric Department carries a heavy responsibility of routine clinical work in wards, in outpatient consultation services, and in laboratories. That part of its work is impelled by the usual interest that now prevails throughout the world in diagnostic and therapeutic medicine. A new physical sign is eagerly seized. A rare disease is exciting news. A miraculous case is the very breath of life to all of us, particularly to the young men who find complete intellectual interest in that type of medicine at that stage of their career.

Wishing to widen our experience beyond the wards and laboratories, we have set aside a team of field-workers who work largely as the research department for the city health authority in social and preventive medicine. It is a two-way process. We present problems to the city authorities, and they bring their problems to us. We did some early surveys in differential growth and development of children in various social groups, but our interest was not

firmly established until the city asked our advice on its rising infantile mortality between the years 1935 and 1938. The health authorities were disposed to counter this by opening more child welfare clinics. But after consultation it was agreed that it would be better to get the real facts on death and disease in the city before making expensive programmes, which may savour of Chapin's advice, 'when the facts are not at hand have the courage to do nothing'. We undertook to organize a detailed and personal investigation, first on a sample of infant deaths in the city, with their relevant familial, economic, social, and clinical features, and later a similar inquiry into morbidity, particularly of the acute infective illnesses as they arise in the homes of the people. The research had the designed purpose of providing facts on which we could advise the city health department about its child welfare programme. The mortality study continued throughout 1939. It was carried out personally by Dr. F. J. W. Miller, with occasional help from me and the part-time services of one clerk. In that year there were 272 infant deaths in the city. We were able to make an immediate and adequate inquiry into all but one of these. Our results were not remarkable, but they gave us hard facts with which to argue, and we were able to reveal the wide disparity between the notified cause of death and the true cause of death, thereby pointing the way to a need for better vital statistics on infantile mortality.

The benefit of this study was mainly to ourselves in giving us experience of methods that involved the quick and spontaneous co-operation of all the doctors, hospitals, nursing homes, and health officials in the city. We acquired a technique. Out of this has grown a companionship that now serves us well. The war interrupted us, and we were not able to resume the second part of it until later.

In 1946 we began to design an investigation of infant morbidity for the city. Its main purpose was to study by direct observation the acute infective illnesses of infants. We wanted to know the incidences, the clinical types, and the results of these diseases studied against the background of the homes and families in which the parents lived.

We had in mind three other long-term purposes. The first was to widen the interests of the young men working in the wards and laboratories, by bringing them in daily contact with the field

searchers. The second was to provide ourselves with relevant local facts wherewith to freshen our clinical teaching and to guide health programmes. The third was to create a method to give us a contemporary picture of disease verified by statistics and direct clinical observation, which would reveal new problems for research.

We spent several months preparing our methods of inquiry. These included meetings with the medical practitioners of the city, whose co-operation was essential; a precise liaison with the public health department, with whom we have kept in step at every stage until they regard it as a joint research; a co-operative arrangement with all hospitals and nursing homes; and a study of methods of entering into the homes in a manner that would make us welcome. In that preliminary period we busied ourselves with assembling a team of workers, and in determining the sample to be studied and the method of making observations and records.

We decided that a sample of about 1,000 infants should be taken and studied from the day of their birth. This represented about a sixth of the city's births in a year. We had the alternatives of picking every sixth child or taking all the births in a sixth of the year. We chose the latter, and decided on all the infants born in the months of May and June 1947. This gave us an easily identifiable sample. From then on we could recognize and easily remember that a May-June baby was in the sample—or a 'red-spot baby' as they came to be known. The choice of May and June was deliberate, having the advantages of allowing us to undertake the initial heavy work at a good season of the year.

The number of infants born in these 2 months was 1,142, representing a complete cross-section of all social groups of the city. Out of this total there were 6 non-co-operators, 2 of whom were doctors. This is some index of the success of our methods of approach. It is necessary here to indicate that the people of the north of England are mainly indigenous, shrewd and independent, suspicious of strangers, and great home lovers. Their successful co-operation was, I think, mainly due to the courtesy of the preliminary explanation given to the parents by doctors whom they knew and trusted. They were promised no rewards or benefits. They understood clearly that we were there only to observe and understand their difficulties.

Samples chosen in this way will vary with the social structure of the city. The structure of our sample is illustrated by Table 18.

TABLE 18
Social class by occupation of father

<i>Class</i>	<i>No. of infants</i>
Professional	32
Black-coated workers	93
Artisan	602
Semi-skilled	172
Unskilled	163
Not classifiable (largely illegitimate)	80
Total	1,142

The incidence of illegitimacy also has an influence on the sample. In ours it was 5.7 per cent. (65 in 1,142). The place of birth was a factor for consideration both for the purpose of comparison and for the aetiological studies of infection (Table 19).

TABLE 19
Place of birth of infants in the sample

<i>Place of birth</i>	<i>No. of infants</i>
Home	655
Hospital	353
Nursing home (private)	131
Not determined	3

After this glimpse of the sample I return to method. We took 5 nurses who were health visitors and trained them in our methods. We knew the sort we wanted, and we got them. They sat in on the preparation of the inquiry. Each of them took about a fifth of the families as her parish. It was their duty to make themselves intimate and familiar with their parishioners or, to put it more pompously, to understand their social anatomy and physiology. The first visit to the home and family was the most important. The nurse then established a relation, and all else followed easily.

By previous experience we determined at what intervals a regular visit should be paid by the nurse, and by a preliminary pilot survey we tested our methods. The technique was that she should make regular visits, gather all the relevant information at each visit, and come again or call when necessary. Apart from the basic information about housing, living, feeding, and working arrangements,

which she collected with as much tact as possible, it was her main duty to collect the details of all illnesses or ailments, however slight, and to bring the doctors in on consultation whenever necessary. The inquiry was in the first place a study of the incidence, nature, and results of infective illnesses. After experiments we found that a mother's memory of the minor ailments of her children was not accurate beyond 7 weeks. On this finding the regular visits were arranged at intervals of 6 or 7 weeks. Also, it confirmed my view that the questionnaire method would not suit our purpose.

The team was completed with 2 clerks, 1 doctor devoting most of his time to the research, and 2 other more senior paediatricians called in for special observations and studies. Each week's collected material was discussed, digested, and collated at a Saturday conference. We brought to this the medical officer of health, his housing experts, and others concerned whenever necessary. The complete records revealed in detail how a thousand families live and have their being, how their children are born and bred, what their ailments are, and the relation of these to others in the family. By direct experience and observation we know what has happened to these children. It has given us material both for social history and for clinical medicine. It started as a study of 1,000 infants. It has become a study of 1,000 families.

By the end of the first year the sample was disposed as follows: 44 had died, 6 had refused to co-operate, 125 had removed from the city, and 967 remained in the inquiry. The 125 removals were more than we expected. They reveal a temporary social difficulty due to a shortage of houses, by which young married couples returning from the war started their married life in lodgings or parents' homes until they removed to houses of their own.

The inquiry has now completed its second year, and we have decided to continue it for 6 years to come, mainly for the purpose of studying the familial pattern of disease, particularly respiratory illnesses and their late results such as bronchiectasis. Its records will be a formidable document, but the statistics will be vital. Already they give a clear indication of how people live, how they cope or fail to cope with various difficulties, how they make use of their family doctor, and what their attitude is to hospitals and health services.

I do not intend to display the clinical and epidemiologic results; I shall choose only examples to demonstrate how these may be

used to give reality to clinical teaching. To experienced practitioners of medicine there is nothing new in what I have to say, but it helps those who practise only in hospitals to broaden their concepts of disease and gives them practical material for teaching.

WHOOPIING-COUGH

The simple clinical facts about whooping-cough are easy to understand, but a clear picture of the disease in all its variations is hard to come by and still harder to demonstrate. The sample survey method gets near to it as our experience shows. Before they had reached the age of 12 months, 100 of our 1,142 infants had whooping-cough. The source of infection was intrafamilial in 41.

Of the 100 affected infants, 12 had severe whooping-cough of the clinically obvious type, and of these 3 died; 65 had whooping-cough intermediate in severity and difficult to diagnose on clinical grounds alone. The remaining 23 infants had the disease with symptoms so mild, lacking whoop or paroxysm, that diagnosis was reached only on epidemiologic or bacteriologic evidence. In these the illness took the form of a respiratory catarrh, with insignificant cough.

In the second year of the survey we saw examples of whooping-cough in infants who had had the disease in the first year. The presumption is either that they had failed to develop immunity from the first attack, or that one of the illnesses was pertussis and the other parapertussis.

This intrafamilial method of clinical investigation gave us a simultaneous view of the disease in other members of the family and showed that whereas most of the older children had their whooping-cough in its classic form, the disease could have been diagnosed with certainty on clinical grounds alone in not more than a third of the young infants affected. A knowledge of its intrafamilial epidemiology and its subclinical variations had to be called into assistance.

Let us look at the use of these clinical and epidemiologic facts in teaching. In the first place they give a topical picture of the disease, which could be continued year by year to demonstrate and calculate the trends of events. Possessed of this contemporary and serial knowledge with statistical confirmation, we can take the second step of evaluating a particular treatment or preventive programme

such as vaccination. In this way the student begins to see a disease from many angles. He is no longer looking at it through the blinkers of diagnostic medicine.

OTHER INTRAFAMILIAL INFECTIONS

One of the main projects of our investigation was to study the different types of respiratory illnesses and their results. While doing this we gain insight into the aetiology of a variety of diseases including streptococcal illnesses, staphylococcal illnesses, and tuberculosis. Here I use aetiology in its literal sense to include the personal and environmental circumstances leading to infection. Working in this way from within our sample is like watching a play from behind the scenes.

Here I can do no more than pick out a few illustrations for general comment. We found nearly 60 of our families in which there was a long history of repeated staphylococcal infections, carrying with it a burden of considerable discomfort and disablement. These families demonstrate how defective medical treatment may be if it is merely episodic, and if members of the family consult different doctors or attend different hospitals. Only a doctor with a personal sense of responsibility for the whole family and knowing each member of it is likely to string together all the episodes of 10 years to demonstrate the unity of the varying illnesses and to take steps to eradicate them, as he might eradicate scabies under similar circumstances. Our findings raise questions both of the carrier state within the family and of familial variations in immunity that may be genetically determined—a branch of serology that has received little study.

The method of investigation illustrated well the variations of disease manifestation according to the patient's age. To illustrate, I quote an example of respiratory infection. In a tenement building we saw a community of 22 ; in 7 families Through this community a respiratory infection over a of 8 weeks. All the 3 babies in 3 different pneumonia, and the other 2 with clinical symptoms: of the children he all respiratory illness of the children and slight catarrhal.

which epidemiology linked to clinical medicine widens our view of a pathologic process in all its variations and brings out well the factor of age in aetiology.

Since our sample is a random selection including all classes of the community it gives us a clear indication of the clinical and social effects of tuberculosis. Regular tuberculin testing is the basis of this observation, and in this way we are getting exact information about the tuberculization of the group, seeing each child at the time of its primary infection and afterwards. In this field the investigation acts as a constantly recording apparatus, pointing out where the danger lies and how it is operating. Even in so well worn a subject as tuberculosis we are being beset by new problems, of which one of the most interesting is the group of children under observation in infective families who fail to become infected. They remain tuberculin negative in spite of the risk. This is raising the question of the secondary factor, which arises to determine the passage of tubercle bacilli through the skin or mucous membranes.

CONCLUSIONS

I cannot enter into more clinical details, and I have restricted my observations to a few illustrations of how field studies combining clinical medicine and epidemiology may be used to give a perspective view of a disease with its clinical variations, its age variations, its epidemiologic vagaries, and its social effects. If they make no new discoveries they tie up a good many loose ends, and out of this experience the investigators will be able to advise on child-health programmes in a clearly determined order of priority. It also uncovers diseases and illnesses difficult to identify or to classify, and thereby poses new problems of research. Finally, it is creating an instrument of clinical observation correlated with exact statistics that gives authenticity to clinical teaching. I am encouraged to believe that John Clarence Cutter would have approved.

I have done no more than suggest how clinical study may be carried outside the hospital and the clinics. My final question must be to ask, Is it worth-while, or is it mere pebble counting? The answer to this is given by my colleagues Dr. F. J. W. Miller, Dr. Donald Court, Dr. Hugh Jackson, and Dr. Ruth Fawcett, who are responsible for the work, and the secretaries and the health visitors who assisted them so skilfully. They have brought together

a wealth of detailed information from which we learn a great deal of clinical medicine and a great deal also about life itself, and the experience has transformed our own outlook on clinical medicine and clinical methods.

Whereas we had previously been concerned too much with diagnostic medicine and with the experimental study of phenomena, this field-work has developed and confirmed an idea that has been in my mind for many years. Too much attention paid to diagnostic medicine diverts attention from the detailed study of the sequence of clinical events that makes an illness. A scientific study of this sequence of events, minutely observed, carefully recorded, and with each event placed in its right time relation, is the method of the natural scientist, and it becomes a powerful instrument in the training of the young clinical scientist. To know the hourly or daily changes in the progress of an illness is clinical science of a different and higher order than mere diagnosis. To be able to recognize the slight but significant changes of typhoid fever that will take place between the seventh and ninth days of the illness requires greater skill than to diagnose the disease. By training in such work we become alert to the recognition of new syndromes or new diseases, and we become aware of the trends of disease in their epidemic fluctuations and clinical variations. Combined with statistically based field-studies we thereby get a wholesome view of clinical medicine.

In the social field the chief lesson I have learned in our studies of disease in families is how well the people cope with their domestic difficulties. Less than 7 per cent. are feckless or happy-go-lucky people who need help if they are to maintain a decent level of care of their children in health or sickness. The remainder are stalwart people coping with life and remaining loyal to their responsibilities.

XIV. *A Thousand Families in Newcastle upon Tyne*¹

THE book, *A Thousand Families in Newcastle upon Tyne*, is a record of an inquiry designed to identify the diseases of childhood in a representative sample of families, to trace their origins, and to measure their effects. The observations were made at the source of the illness, in the homes of the children. Other relevant information and observations added thereto include a description of the families, their homes, and other circumstances in which they lived.

The observations cover the period from the birth of the children to the end of their first year, but the inquiry is designed to continue for 7 years and perhaps longer, so this report is to be regarded as a first instalment to be followed by others which will probably be in the form of monographs giving the results of particular studies in subsequent years. The inquiry may thus end in an accurate and perspective picture of disease in childhood which will be useful not only to medical practitioners and to public-health officers, but also to medical students in their search for clinical education. Through references which have already been made to the research in preliminary papers and lectures it has come to be known as 'The Thousand-Family Survey'.

In designing the experiment we decided that in no other way could we obtain the facts we sought than by an intimate and continuous study of an adequate sample of the families in the city. It was to be primarily a clinical and epidemiological study substantiated by direct observation and immediate record. We knew the difficulties of such an experiment. It would require loyal co-operation between the research team and the family practitioners. It would demand great patience. It would eschew the questionnaire. It would depend upon the adherence of our sample families who might otherwise break away and so invalidate our statistics. However, from previous experience of similar work, we thought that the city of Newcastle upon Tyne was well disposed to help us over these difficulties.

¹ From Spence, J. C., Walton, W. S., Miller, F. J. W., and Court, S. D. M., *A Thousand Families in Newcastle upon Tyne*, London, Oxford University Press, 1954.

We had gained experience of this kind of work in a survey of deaths in infancy in 1939 and we were busy with the extension of this inquiry in a study of morbidity when the war broke out. We postponed our work and resumed it in 1947. By that time there were additional reasons for surveying and estimating the range of illnesses in infancy and in childhood. Records kept for us by general practitioners showed that one-quarter to a third of their work was concerned with sick children, and that only 2 or 3 per cent. of these children were referred to hospitals. This denoted a large variety and volume of illnesses of which the hospitals had little experience, much of it concerned with ill-defined infective diseases of the respiratory organs. It was towards these illnesses that we intended mainly to direct our researches, by carrying them beyond the hospitals, where the scope of observation was limited, into the homes of the people, where the view of disease was wider.

Surveys and inquiries which make an intrusion into family life demand a justification beyond the mere satisfaction of curiosity. They can be justified only if they are designed to answer questions which are worth answering, which have not been answered before, and which cannot be answered in any other way. Otherwise they slip too easily into the repetitive enumeration of trivialities. But they demand a prior justification in the approval of the families and their family doctors, and this can be vouchsafed only after full explanation of their purpose. We took preliminary steps to ensure this approval. In all that followed we tried to remain sensitive to the privilege of access to the homes of our families, and we have been scrupulous in avoiding anything which might interrupt or mar the relationship between a family and its family doctor. The harmony of our arrangements can be judged from the fact that we have had only four desertions from the sample.

The arrangement of the report is straightforward. The investigation was an experiment in organization and technique, so we first describe its origin, purpose, and method. Local in character, it should be seen in the context of the conditions prevailing in Newcastle upon Tyne in 1947. The chapters in Part I describe the design of the investigation and the selection of the families, and give a brief account of the history and character of Newcastle and the development of its health services. Part II is concerned mainly with our clinical observations and findings. It starts with a résumé of mortality and morbidity in our survey-group as a whole, and in

successive chapters each type of illness is considered separately and in detail. Part III records our studies of the environments which influence the health of children, and in it chapters are devoted to housing, family structure, maternal capacity, infant-feeding, prematurity, illegitimacy, and the medical care of children. Finally, Part IV summarizes the year's work and our suggestions for its applications. The statistical details are presented in the appendixes.

We are often asked about the cost and organization of the survey. At a time when so many schemes are in the air to promote research into social medicine and to provide buildings for this purpose, it may be worth recording that the survey has been carried out by the existing staff of the Department of Child Health, King's College, Newcastle upon Tyne, and the Health Department of the city, with the additional help of one research worker provided in one year by the Medical Research Council and in subsequent years by the Nuffield Foundation. As the work consisted mainly of field research, the only special accommodation we have required has been two small offices.

We know of four other investigations which, although different in kind, can be related to our study. In Western Reserve University, Cleveland, U.S.A., Dingle and his colleagues are engaged in a detailed and intensive study of respiratory disease in 57 selected families in an attempt to throw light on fundamental problems of infection and immunity. The Population Investigation Committee has instituted a national survey collecting information from 424 maternity and child-welfare authorities about the children of 14,000 women (*Maternity in Great Britain*, London, Oxford University Press, 1948). The Luton survey is concerned with morbidity in childhood in an English town of good economic standing; though it carries the impediments of retrospective inquiry its results are comparable with ours (Dykes, R. M., *Illness in Luton*, Luton, Legrave Press, 1950). The late Professor J. A. Ryle and his colleagues in the Institute of Social Medicine, Oxford, have studied the development of children in volunteer families; this study will not reflect the community but it will add new anthropometric facts. Each of these investigations is designed to answer different questions, and together they should extend our knowledge of health and disease in childhood.

In carrying out our own survey, the extension of knowledge has not been our only, or perhaps our predominant, motive. We have

had in mind also the need to extend our own experience to enable us to teach more clearly and more cogently about illness as it arises and shows itself within the family. We have had in mind also the prospect of adjusting the invaluable experiences of health visitors or child-health nurses to the work of family doctors.

CONCLUSIONS AND SUGGESTIONS

The gathering of facts is not everybody's meat, so we turn finally to some conclusions and suggestions arising from an experience enriched by close companionship with the staffs of public-health departments and with family doctors. In this collaboration with other branches of the profession we could not have wished for a more harmonious identity of interests.

The pertinent facts out of which our suggestions will arise are as follows:

1. The remarkable decline in disease and deaths in infancy during the past 40 years is due primarily to an increase and spread of the knowledge about the proper care and feeding of infants, and to a decrease in poverty which has resulted in an amelioration of conditions under which people live.

2. The chief agents of these improvements have been (a) those mothers and grandmothers who have sought this new knowledge about the care and feeding of children and applied it in their families; (b) the child-welfare centres, the health visitors, the newspapers, the radio, and other organs of publicity which have spread this knowledge in their different ways; and (c) the public authorities and others who have built new houses.

3. While the family doctors and the hospitals have played an essential part in the diagnosis and treatment of established disease, their work has not been primarily responsible for the improvement in child health in the past 40 years, mainly for the reason that medical education did not, in that period, equip medical practitioners for a role in preventive medicine.

4. The universities have contributed to the improvement in child health only in so far as some members of their staffs, by their researches, have advanced or clarified knowledge of nutrition and of disease in childhood. Until recently the university contribution to the teaching of this subject has been slight.

5. In spite of the improvements in child health during the past

40 years, there still remains a considerable volume of disease in infancy which is preventable, and beyond that a further volume of disease which, although not preventable, is curable, and will thereby make demands upon research workers for its clearer understanding and on trained medical practitioners for its effective management.

6. There is a limit to the results which the traditional agents of housing, health education, and sanitary reform can achieve in the further prevention of disease in childhood, and beyond that limit they will be subject to the law of diminishing returns. Further prevention will now depend mainly on the education of family doctors in the principles of health and development of children and in the prevention of their diseases, and afterwards in the application of their knowledge in their family practices. This work will be undertaken the more effectively if they co-operate with the traditional agents of preventive medicine.

7. An extension of the concept of family doctoring will depend also on a greater knowledge of disease in infancy. In order to apply this knowledge, an improved clinical education is required for those men and women who will become family doctors. This improvement in clinical education presupposes that the teachers themselves will base their teachings on clinical research and field inquiries which will indicate where the need for knowledge is greatest and how it can be applied to the local scene. This throws back on medical schools the responsibility for a better clinical education and a more realistic training of undergraduates and graduates.

The facts which we have enumerated indicate that the time is now ripe to alter and reform the various institutions which maintain the health of children and prevent or cure their diseases. Our final suggestions concern these alterations and reforms, and they are based on the assumption that the primary responsibility for child care will remain with the parents, and that professional institutions will do nothing to diminish that responsibility but will act as aids to parents in what is one of the most necessary and satisfying of all human activities.

First suggestion—medical education

Medical education in this country is bedevilled by the fragmentation of its curriculum, by the irrelevance of its arrangement, by

its examination system, by the authority of extramural professional bodies who can impose their will upon the universities, and by the tendency of teaching hospitals to lose their facilities for the undergraduate education of medical students and to become places for the training of specialists.

Educational reforms do not come from committees, councils, or conferences. They arise only from experiment, proof, and example. Our suggestions, therefore, go no farther than these—that universities should experiment in arranging their undergraduate medical curriculum in various ways, one of which should be to shorten it to not more than 4 years; that examinations should be designed and used to test the methods of education and not as ends to which the education is directed; that the last 2 pre-registration years of a student's practical clinical training should be free from the interference of examinations in preliminary and non-clinical subjects. With this freedom experiments in practical clinical training could be designed by teachers who have a clear idea of what a family doctor's work is and should be, but it is not likely that a new design of practical clinical training can take shape until these facts have been collected by inquiries and experiments.

If the reader turns to the chapters in *A Thousand Families in Newcastle upon Tyne*, on the frequency of illness (Chapter V), and the serious but infrequent diseases (Chapter XIII), the paradox of clinical education is illustrated, and although the facts in those chapters are concerned with illness in the first year of life as seen by family doctors, the principles involved apply equally to their other fields of work. On the one hand there were about 1,400 infective illnesses nearly all requiring precision in diagnosis and treatment, but few of which are studied or presented for teaching in hospitals; and, on the other hand, there were the 18 illnesses such as intussusception, meningitis, and osteitis which are the classical material of teaching hospitals. The medical schools must face this question of how to present a balanced view of disease to students in training, and how to give them experience of the illnesses they do not at present see in hospitals. It is no easy task, for it carries the responsibility also of teaching about the rare but serious diseases which a family doctor is expected to diagnose although he may meet with them only about 5 or 6 times in a lifetime of practice.

Our survey has shown us one method by which clinical teachers

of paediatrics can regain this perspective. In a more formal way this kind of field-work could develop into a sub-department of clinical epidemiology within the department of child health or paediatrics, where it would operate, however, not within the hospitals but by field inquiries which would keep in touch with family practitioners.

Another practical suggestion is that the children's wards of infectious fever hospitals should be linked to the teaching departments, and that when new children's hospitals and departments are built, wards for infectious disease should be incorporated in them, thus following the example of many continental and American hospitals. Most of the illnesses of childhood are due to infections. The old infectious-diseases hospitals dealt only with a few aspects of these infections. The children's hospitals and departments deal with other aspects. Their forces should be combined in new types of children's hospitals. Unless that combination takes place, research and teaching will remain distorted.

The advance of knowledge about disease in childhood has been so rapid in recent years, and is continuing at such a rate, that regular post-graduate teaching of family doctors has become a great responsibility of the universities. Twenty years ago it was possible for a man to enter practice without any instruction about disease in infancy. Even today that instruction may not extend beyond a few demonstrations and lectures without opportunity for more than occasional practical experience. The practitioner feels this lack mainly in his use of modern methods of treatment in the many infective illnesses of infancy. Without precise postgraduate instruction in the use of these methods, he is left at the mercy of advertisements which come to his notice more easily and more quickly than textbooks or articles in medical journals. This brings us back to a principle of teaching of men and women who are family doctors. The teachers must first inform themselves of the content and conditions of family practice.

A few words only about research, which is mainly an affair of the universities. Disease in infancy is a comparatively new field of medicine waiting to be explored. Although this is mainly the work of the universities, the local health authorities and staffs of hospitals must look to research for their guidance. The mere clarification and interpretation of facts about routine work is itself a kind of research, and it appears to us reasonable that a major local authority

should devote a sum equivalent to 5 per cent. of its total annual expenditure on child welfare to research of this kind, and seek the advice and help of the university in arranging the inquiries and investigations on which the money should be spent. It would be a wise economy to have such expenditure on public health and social welfare guided regularly in this way.

Second suggestion—family practice

We have no wish to speak beyond our knowledge, but there is one observation which has grown stronger with each year of this investigation and which is relevant to the development of general practice in this country. Infection in the first year of life is almost always an expression of family infection, and yet we were surprised how rarely the doctor, the health visitor, the specialist, or the parents appeared to consider the problems of health and sickness in family terms. The natural inclination is to seek and to find an isolated disease in an individual patient. A family approach, on the other hand, the value of which has been increasingly impressed on us in the past 6 years, would modify medical practice in several ways. First and foremost it would become fully accepted that the intelligent care of children requires one doctor for the whole family—requires in fact a family doctor. This leads us naturally to reject the view, which is growing in the United States, that the home care of children should be in the hands of separate paediatric practitioners. We feel that such a development would not be in the best interests of family life and domiciliary practice in this country. We recognize that our family doctor, dealing not only with the home care of the sick child, but helping the parents with the many problems of growth and behaviour peculiar to childhood, will need a wider understanding of child health than he has at present. Already nearly a third of the practitioner's time, and far more of his concern, is devoted to children. We would recognize this as a field of medicine which falls within his proper responsibility and we would invite him to join with us in a continuing process of mutual education which would make specialist and family doctor alike better equipped for the care of children. The ways in which this can best be done can be no more than mentioned here. A wider use of consultation together in the home, the more frequent discussion of a doubt or a problem over the telephone, the sharing by a practitioner for, say, a week in the ordinary work and conversa-

tion of a children's department rather than a pretentious refresher course, the use of local news-letters to bring rapidly to his notice current advances in diagnosis or treatment, are some of the ways in which the isolation of family practice from hospital and hospital from family practice could be overcome. The family doctor in this old yet modern sense will always be the chief agent in whatever supporting services we offer to the parents. He can, however, no longer meet the need alone, and we must now consider what his relationship should be to the public-health services which have developed during this century, and what additional help he requires to cope successfully with the children and the family life of his practice.

Third suggestion—family doctor and local health authority

We accept as axiomatic that family practice cannot be conducted entirely by the individual effort of a doctor working alone, and that one of the family doctor's most important relationships in the conduct of his practice will be with the local authority, so that he may know how to use its resources.

The work of the local authority is entering a new phase. Having passed through the stage of environmental hygiene and the direct provision of personal services, it must now provide and administer home nurses, health visitors, midwives, and may provide home-helps, and make them available to those doctors who wish to take advantage of them. If the local authorities rise to this opportunity, and if the family doctor makes use of this help, he will find that he is brought into active relationship not only with the district nurse and midwife but also with the health visitor and the children's officer. These are the chief field-workers of the local authority. They are institutions ready for the family doctor's use, and he should understand who they are, what they can do, and how they can help him in his care of his families. He must be willing to use their services with economy, with appreciation of their experience, not to do the work he should do himself, but to supplement his advice or to investigate further into a situation.

Here we are concerned with his use of the health visitor. She could help the family doctor in his supervision of infant-feeding, in the nursing tasks of infancy, in the hygiene of the child which may go astray to produce disabling symptoms, and in giving help with the nutrition of older children. The more she is used directly

by the family doctor the more effective will be their joint efforts. She can not only help the doctor but also enhance his responsibility and his authority. He will gain a useful ally and she will regain point and purpose in her work.

Surveys of general practice are usually only made from the evidence of doctors, and this shows how little of this co-operation exists. The fault is partly with the doctors and partly with the health authorities. When this is adjusted, co-operation should arise as opportunity allows, and, although carefully fostered, its growth should be natural. It might be set back by attempts at formal agreements. It would grow unequally in different areas, but examples of good co-operation would doubtless be given to point the way. Out of this experience some doctors might develop their own arrangements for antenatal or child-welfare work in which the health visitors could assist. It is by growth of this sort that we hope to see the slow merging of the work of health visitors with that of family practice.

Fourth suggestion—local health authority

During the years we have worked with our health visitors in the survey we have seen how their enthusiasm can be aroused, and we have realized the wealth of social and medical knowledge they possess and exercise in the course of their work. We feel, however, that this enthusiasm and the full use of their particular skills can be maintained only under certain conditions.

The effective work of the child-welfare services as they exist at present depends largely upon the activities of health visitors in homes and welfare centres, and to a much lesser extent on the services of doctors in the welfare centres. These centres have a record of valuable work, but conditions are changing. Problems are changing with them, and the time has now come for a reassessment of the work of the child-welfare services. The first step should be to extend and alter the work of health visitors to make it a more satisfying and therefore a more effective occupation. The following are suggestions on how this might be done.

1. The health visitors as a body should be brought regularly into conference with the Medical Officer of Health and be more closely identified with the formulation of his policy. We know the value of that from our experience of co-operating with health

visitors who shared in the original design of techniques for the survey.

2. Health visiting is physically tiring and may become a monotonous task, unrelieved by praise or professional companionship. Health visitors should be identified with a district and with the family doctors of that district. In that way co-operation and professional companionship will be established.

3. Health visitors should not attend families in their homes without definite point and purpose. They should not visit merely for regularity or records. Much of the continued visiting after measles and other infections could now be abandoned.

4. The health visitor should, in her work, use her hands as well as her heart and mind. She should become a children's home nurse. This, we believe, is possible without losing sight of the need for preventive work and constant education. But it would bring the health visitors into a far closer and more receptive relationship with her families.

The child-welfare centre with its regular sessions is the other great pillar of child-welfare work. We think it will remain so for many years, though its work may alter and become more broadly educational and social. But here we are considering only its function of educating mothers in the care of children. We know it is the better mothers from the artisan homes who go to welfare centres, and at a good centre there is much for them to learn. But they do not need to be visited often in their homes. By selecting her families the health visitor could devote more time to those families who do not come to the centre and who they know need help. The work of the welfare centre and of home visiting would then be more truly complementary.

The medical staff in welfare centres will vary from place to place. A centre with a keen, interested doctor is a very real focus of education for young mothers, possibly with an authority never attained by the health visitor alone. A welfare centre with an indifferent doctor is dull and ineffective. While some centres could be staffed at some sessions entirely by health visitors it is doubtful if it would be good for all welfare centres to be so. We do suggest, however, that the doctors should not be wholly engaged in welfare-centre work and should have other medical responsibilities in family or hospital practice.

Finally, in university cities or near to teaching hospitals, we

would like to see the welfare centre play a far larger part in the teaching of child health, for there the student can see yet another aspect of life and family practice.

Fifth suggestion—economy of co-operation

Our final suggestion is that in each local area of appropriate size, as, for example, a county borough and its neighbouring or adjacent boroughs, a method of regular consultation between all parties concerned in child health should be created. This could take the form of a children's medical-care council, but to give it reality it must be invested with powers. It should be a working party and not a periodic congregation of separate interests. As a working party it must be supported by research and inquiry. Its inquiries would uncover outmoded institutions and demonstrate reforms. For this work it would recruit professional aid and advice, remembering that research is a professional affair. Its power would come from the force of its opinions clearly announced. Its purpose would be a true economy of institutional effort, using these words in their literal sense of putting one's house in order. To make it representative it must include those who can speak for parents, for medical practitioners, for hospital services, for the child-welfare services, for the school medical service, and for the housing authorities.

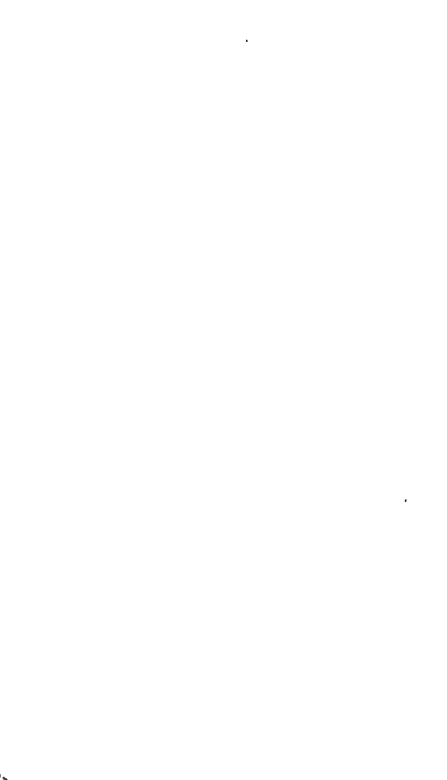
This group of citizens and professional people would establish criteria on which to form their judgements. They would know the significance of mortality and morbidity rates. They would follow the trends of disease. They would be informed about methods of hospital care and about methods of after-care of children who have been in hospital. They would review the changes which are necessary from time to time in the organization of nurseries and child-welfare services. Education and enlightenment is more likely to come about through such a body than through the impersonal national conferences which are promoted at great inconvenience and expense. We consider that the need for such a council is not less important for the care of sick children and the prevention of disease than is a committee for deprived children. The committees for deprived children have already found an important function. We have no doubt that local children's medical-care councils would be equally valuable and effective.

Out of the experience from which we draw these conclusions

and make these suggestions we are left with the impression of a great number of authorities, institutions, agencies, societies, and professions each taking its share in arranging the health and welfare of children. They recognize that the final responsibility rests with the parents, but often they are at a loss to know how to bring their aids and encouragements at the right time to the families which need them. Fortunately more than 80 per cent. of the families can manage their own affairs if they have a house to live in, and a good neighbour, a nurse, a school teacher, a priest, or a doctor to come to their aid when necessary. But in the nomadic societies now to be seen in some industrial towns these relationships no longer exist. To re-create them, in this changing scene, will require a synthesis of understanding in the minds of all who take part in these services. This understanding must begin in a knowledge of the facts.

PART IV

The Care of the Sick Child



XV. *The Care of Children in Hospitals*¹

THE forms of human institutions are predetermined as much by the age in which they are set as by the men who set them. Charles West founded the Hospital for Sick Children in Great Ormond Street in 1851. In this instance the age overcame the man. He was an obstetrician of the restless reforming type, as became the son of a nonconformist minister who was half pedagogue and half evangelist. Through his father's school, through an apprenticeship to a local apothecary, through St. Bartholomew's Hospital, and through several continental universities he pursued a notable academic course with serious pertinacity. Afterwards he practised midwifery in London, with a reputation and success which made him Lumleian Lecturer, Harveian Orator, and Senior Censor of this College. Near the age of 40, which is the climacteric of the reformer, he turned his energies to propagating the study of disease in childhood in lectures, books, and pamphlets. He was obsessed by the appalling ignorance in this branch of medicine. That more than one-third of the children at whose births he attended would die before the age of 12 weighed heavily on his mind. He conceived the idea, rightly or wrongly, that the quickest remedy was to open a children's hospital which, he said, 'would afford the means of instruction to students, as well as furnishing opportunities for extending the boundaries of our knowledge concerning a class of diseases frequent in their occurrence, dangerous in their character, and often obscure in their symptoms'. Against much opposition he succeeded in founding his hospital, in the house which was already famous as the residence of Richard Mead, and there he worked for 23 years. But, as Norman Moore said of him, 'the conduct of other men so rarely satisfied him that he was not a happy colleague, and left both St. Bartholomew's and the Hospital for Sick Children in a state of feud with other members of the staff'. His remaining years were spent, as is the way of such men, in brooding over medical education and in other discontents. So much for the man whose name we honour in these lectures.

¹ The Charles West Lecture, delivered at the Royal College of Physicians, London, on 19 November 1946. Published in *British Medical Journal*, 1, 1947, p. 125.

of admitting children because of the danger of death from cross-infection. The early children's hospitals avoided this difficulty by refusing to admit children under the age of 2, which, although a justifiable procedure at that time, was an ostrich-like policy towards study of disease in early infancy. Therein lies a partial explanation of our comparative ignorance in this most difficult and least understood branch of medicine. Charles Cameron's remark still holds true: 'There is no branch of medicine in which experience is more necessary and yet more difficult to come by.'

A discussion of these problems is not an academically exciting subject for a lecturer. It is difficult to raise it above the level of a pedestrian guide, but I choose it for three reasons. The first is its practical importance at a time like this, when the planning of new children's hospitals is in the air and when their special needs may again be subordinated as they were a hundred years ago. The second reason is that the problems involved are particular and technical, and I wish to suggest that they cannot be understood unless the facts are carefully analysed. The third reason is that during the past 2 years, as a member of the Curtis Committee, I have had the opportunity of examining many kinds of children's institutions, assessing their methods and comparing their results, and this experience has taught me that the care of children is as uneven in our hospitals as it is in other institutions—good in some and very bad in others, and where it is very bad the explanation lies usually in a failure to define personal responsibility, or in the imposition of a personal responsibility by a remote governing or administrative body on a staff who fail either to recognize their difficulties or to confess them.

ADMISSION TO HOSPITALS

The number of children's beds required in hospitals is influenced by many factors apart altogether from local variations in the incidence of disease. My observations have led me to estimate that in two children's hospitals or departments both doing the same kind of clinical work the number of beds required may vary by as much as 100 per cent., according to the manner in which the hospital's work is carried out. A hospital with satisfactory working arrangements will deal with as many children in fifty beds as another hospital with unsatisfactory working arrangements will deal with in a

The age he worked in needs little description. The mere mention of it raises pictures of self-confident men and women toiling earnestly in every field from poetry to politics. In many respects it was a great age, but it was an unfruitful time for this experiment of opening children's hospitals because the strongest interests were then flowing in other directions. In medicine they were towards sanitary reform or morbid anatomy. Elsewhere they were centred on religious questions and on the self-discipline of character. In practical affairs the puritan ethos was being harnessed to iron, coal, and railways in new industrial enterprises, in a prevailing atmosphere of excessively bad taste which permitted even the dons of Oxford and Cambridge to erect architectural monstrosities. That Charles West and his colleagues achieved so much in circumstances so adverse was praiseworthy. They worked under difficulties. Their hospitals bore the impress of these difficulties, and we are still affected by them.

The chief of the adverse circumstances which beset the founding of the early children's hospital was the established authority of the adult hospitals already in existence for more than a hundred years. Man being mainly an imitative animal, it inevitably followed that children's hospitals were built and arranged like the older hospitals. They inherited the same oblong wards and cheerless corridors. They imitated their methods of staffing and of nursing. They pursued their methods of research. This was a misfortune, because the needs and arrangement of children's hospitals differed then, as they differ now, from those of other hospitals. We have not yet escaped the influence of these false conceptions.

Another adverse circumstance that made the eighteen-fifties an unfortunate period for the founding of children's hospitals was the prevailing sentimentality of the times. In this attitude of mind children's hospitals were regarded too much as *refuge homes* for slum children and too little as places for the scientific study of the diseases which might best be treated there and of the methods by which they might best be nursed. They suffered from an overdose of Shaftesbury and Dickens. The paramount need of the time, so far as it concerned the health of children, was a clear analysis of the facts about disease in childhood adduced by those methods of study which, from Sydenham to Thomas Lewis, have revealed so much about the diseases of adult life. Another handicap to the scientific study of children's diseases in hospitals was the dread

policy was the ready admission of children with scarlet fever to the fever hospital, and of children for tonsillectomy to another hospital.

The inquiry covered all children in the ages between birth and the end of the twelfth year. The hospitals and nursing homes drew patients from a wide area, but only those children from the city itself are included. It was estimated that within these ages there were resident in the city 49,800 children in 1943 and 50,400 in 1944. The error of computation in these figures is such that 50,000 can be taken as average for the two years and used as a basis for calculation. The total population living within the city in these two years was estimated at 260,000. Of the 50,000 children, 3,782 were admitted to hospitals and nursing homes in 1943, and 3,704 in 1944. This is an average annual admission of 3,743 (7.48 per cent. of the 50,000 children). Of these, 890 were admitted for tonsillectomy. Excluding the tonsillectomy cases 2,853 were admitted (5.7 per cent. of the 50,000 children). Had the age-groups of 13 and 14 been included, the percentage of the city's children admitted to hospitals would have been slightly lower, as the admission incidence of the older age-groups is lower than in the younger age-groups.

Tables 20, 21, and 22 illustrate the nature of the circumstances under which these children were admitted to hospital.

A perspective view of the various diseases for which children enter hospitals, and of the arrangements needed for their treatment, can be obtained only from a detailed analysis of many factors, including the age of the patients, the season of admission, and, in the case of trauma, the peak hours of the day when the accidents take place. I will quote only a few illustrative facts.

TABLE 20

Types of hospitals admitting 3,743 children

	<i>Patients</i>
Children's hospitals and children's departments of general hospitals	2,163
Fever hospitals	1,218
Children's wards in special hospitals	237
Children's sanatorium	30
Private nursing homes	95

TABLE 21
Ages of patients

<i>Age</i>	<i>No. of patients</i>	<i>No. of patients, excluding tonsillectomy</i>
Under 1 year . . .	419	418
1 year	256	252
2 years	345	312
3 "	338	249
4 "	354	235
5 "	372	261
6 "	365	237
7 "	310	197
8 "	249	166
9 "	227	163
10 "	188	136
11 "	176	127
12 "	144	100
Total	3,743	2,853

TABLE 22
Admissions in age-groups

	<i>Total</i>	<i>Under age 1</i>	<i>Under age 5</i>
Admissions, including tonsillectomy . .	3,743	*419 (11.2%)	1,712 (45.7%)
Admissions, excluding tonsillectomy . .	2,853	418 (14.6%)	1,466 (51.4%)

* 107 of these were treated in infectious diseases hospitals.

Table 23 shows the chief categories of disease, excluding tonsillectomy patients. The distortion of statistics which comes from considering the records of one hospital only is revealed in the fact that, of 761 city children admitted to one children's hospital, 552 (72.5 per cent.) were patients sent for tonsillectomy, while of 962 children admitted to another hospital only 63 (6.5 per cent.) were sent for that purpose.

This enumeration of patients and their diseases in Table 23 shows that the commonest children's diseases are trauma, acute infections, and tuberculosis, but reveals little of what goes on in children's hospitals. It gives no clue to the work of the staff or the arrangement of their duties. We may see the matter more clearly, however, if we examine their duties in three categories. The first is

TABLE 23
Chief categories of disease

	No. of patients	Remarks
Trauma	306	55 had serious burns and scalds, 77 fractures, and 56 head or other serious general injuries.
Abscess, cellulitis, and skin sepsis	202	
Acute lung diseases	312	150 had pneumonia
Tuberculosis	129	3 had bone tuberculosis, and 26 tuberculous meningitis or miliary tuberculosis.
Emergency surgery	79	31 were under the age of 3, and 42 had appendicitis.
'Planned surgery'	115	60 were treated for hernia, and 23 by orthopaedic operations.
Specific infectious fevers . .	929	595 had scarlet fever and 190 diphtheria.
Acute infective gastro-enteritis .	122	
Dysentery	90	
Meningococcal meningitis . .	22	
Rheumatic heart disease . .	15	
Veneral disease	7	
Nephritis	11	
Coeliac disease	7	

the *clinical responsibility* of doctors and nurses in the admission of each child, in its immediate diagnosis, and in its nursing care and treatment. The second is the *social responsibility*, for which I wish I could find a less tarnished phrase to describe duties which involve, amongst other cognate things, the explanation and advice given to parents at the various stages of their child's illness. The third is the *supervisory responsibility*, which devolves on one or more experienced members of the staff in controlling infections, in arranging and rearranging the right juxtaposition of patients on physical and psychological grounds, in adjusting the proportion of nurses to patients and patients to nurses, and in other duties which seem to be more important in children's hospitals than in those of adults. I shall attempt to illustrate these needs by examples, but we may get a clearer view of the picture if we attune ourselves to it by imagining in each case that the patient is our own child. I have yet to find that what is not good for our own children is likely to be good for other people's children—a useful rule of thumb in all matters concerning the care of children in hospitals.

He dislikes the pallid immobile child in the next bed because he is too young for companionship or too ill for talk, but, as is the way of children, he makes the best of it, and carries on a conversation with a boy of his own age 10 yards away over the heads of a whimpering baby and a plaintive 2-year-old standing behind the bars of his cot clad in a shapeless nightgown with a loose napkin sunk to his ankles below. *This young child's plaint is not difficult to interpret.* He draws the attention of a nurse busy with noughts and crosses on a temperature chart. She acts quickly, and then goes to other duties in the kitchen, where she floats mashed potatoes on plates of liquid mince. The children await their dinner, but are distracted by strange events. A white-coated young man arrives and descends upon the silent occupant of a bed who, knowing that her penicillin hour is at hand, breaks her silence in a 4-hourly scream. There are other distractions at other times—the daily or twice-weekly promenade of an older man in black with a retinue of followers; the occasional quick incursion of a younger man more sprucely clad, who pronounces his decision with a 'put him on the list for next Tuesday'; the solemn visit of the matron, who passes from bed to bed with the same question on her lips at every bed; the arrival of an injured child at night; the piece of chocolate after dinner; the excitement of strange instruments which the doctors and nurses use but do not explain. Night comes on, but there is no bedtime story, no last moment of intimacy, no friendly cuddle before sleep. The nurse is too busy for that, busy with the noughts and crosses. This daily rhythm of anxiety, wonder, apprehension, and sleep is better than it sounds, because it is made tolerable by the extraordinary resilience and gaiety of the children at every opportunity. Their cheerfulness keeps on breaking through. But it is a deceptive cheerfulness.

In the hospital there are other wards like this, with a kitchen, a side-room, a linen cupboard, and an entrance corridor beyond which parents shall not pass. They have no treatment room, no laboratory, no accommodation for parents, no interviewing room. Each ward is under one black-coated man of authority, who, although devoted to his work, must delegate much of it to a white-coated resident. He has little time for companionship with his colleagues except in committee-rooms. His ward is his domain. If he is a surgeon it is a surgical ward. If he is a physician it is a medical ward.

Not all hospitals are like this. Some are better, but many others are worse, mainly because most of the clinical work is in the hands of people untrained in paediatrics or insensitive to the fears of children. But I have drawn this picture in order to make concrete suggestions for its improvement.

SUGGESTED IMPROVEMENTS

1. The clinical unit should be big enough to carry a trained nursing and medical staff sufficient in numbers for its varied duties, and working closely enough for the cross-fertilization of each other's minds in daily contact over their patients. For this purpose the unit should contain not less than fifty and not more than a hundred beds, which will be subdivided into smaller nursing charges.

2. The unit should be constructed not as isolated medical and surgical wards but as a combined clinical unit carrying both medical and surgical patients, in which paediatric physicians, surgeons, and specialists combine in their clinical work. In this combined clinical unit the children are placed in rooms or wards on grounds of their age, their temperament, and the nature of their illness, not divided into 'medical' or 'surgical' cases according to their need for operations. I shall return to this theme and its many advantages to patients, to nurses, to residents, and to medical staff. Having worked under this arrangement for many years, I and my colleagues, who have had a full experience of it, are convinced of its absolute value.

3. The unit should be arranged in rooms of five to eight beds, with at least one two-bedded room where, for special clinical reasons or for a companionship which is psychologically necessary, two children may be lodged. In addition, single rooms will be required for 5-10 per cent. of the total number of patients.

4. Conveniently near to each unit there should be a suite of special rooms in which, if necessary, mothers may live with, nurse, and care for their own children. She will do this under supervision of the trained staff. This special mothers' nursing suite should be self-contained and arranged as a domestic flat of five to ten rooms.

5. In each unit there should be a small self-contained traumatic department into which the injured child may be immediately received and treated.

6. In each unit there should be a treatment room in which all dressings, lumbar punctures, and other painful manipulations can be carried out, and where anaesthesia will be frequently used, particularly for the painful removal of dressings.

7. Each unit should contain its own laboratories in which the clinical staff can work and carry out such immediate laboratory examinations as are within their province. One skill enhances another skill. The clinician who has undergone a laboratory discipline and who himself continues, so far as he can, to use precise laboratory methods in his wards is likely to be a more accurate observer and a closer student of disease than he otherwise would be.

8. Each whole-time worker in the unit, whether he be house-physician or senior member of the staff, should have in or near the unit 'a room of his own', however small it may be.

MEDICAL STAFF

The care and treatment of children in hospital demands from the medical staff more time and more attention to detail than does the care of adults. Therefore the primary responsibility for the immediate clinical work in a children's unit should be placed fairly and squarely on the shoulders of one person. In my opinion this should be a resident paediatric physician who has been fully trained before taking up the post, and who will hold it for 2 to 4 years. It is exacting work, and 2 to 4 years is long enough for that kind of responsibility. The resident paediatric physician will require the help of other residents, one of whom will be experienced enough to be his deputy. In a busy clinical unit of fifty beds there should be at least three residents. There should also be a senior paediatric physician and his deputy who visit regularly and who are readily available for consultation. They will be responsible for ultimate decisions, for the maintenance of standards in the nursing and treatment of the patients, and for the promotion of inquiries into all aspects of the clinical work, from the control of ward infections to the control of admissions. In a teaching hospital, where there are associated consultation duties, at least one of the senior physicians should be a whole-time member of the staff. Other visiting physicians, surgeons, and specialists should be members of the staff and visit regularly or be called in consultation.

This method of staffing is designed for a combined clinical unit,

the value of which I press, because it is in such a unit that surgeons and specialists can most confidently place their patients, in the knowledge that the post-operative progress will be constantly supervised. In teaching hospitals and departments there are other compelling arguments for the combined clinical unit. It brings together in close consultation physicians, surgeons, and specialists over the large number of patients whose diagnoses are in doubt. It has the reciprocal action of enlisting the interest both of physicians and of surgeons in new subjects for research. It solves the problem of where to place the 'no-man's-land' diseases such as general injuries and staphylococcal septicaemia with osteitis. It gives to the residents a wholesome clinical experience in differential diagnosis and the after-care of patients. No paediatric physician can practise successfully unless he be an authority on the diagnosis of appendicitis. No children's surgeon can confidently advise operation without considerable skill in the differential diagnosis of acute medical diseases, or without a knowledge of the use and misuse of modern methods of resuscitation. They will best get this experience and skill in a combined clinical unit of not less than fifty beds.

This staffing of a children's clinical unit may sound extravagant. But is it so? Each town with a population of 250,000 will need a general children's hospital or department of 100 beds, an associated hospital for infectious diseases of the same size, and a long-stay hospital of more than fifty beds. A town of that size will be spending not less than £500,000 a year on the public education of its children, and £40,000 or more a year on its child welfare services. Compared with these vast sums the amount which would be spent on paediatric physicians and surgeons is very small. But it must not be *too* small. We must escape the poverty-stricken attitude of a hundred years ago which still encumbers us. We must remember that a trained hospital resident now costs less than a policeman or school teacher.

NURSING

In children's wards the patients and medical staff are in the hands of their nurses to a precarious extent. Rarely in an adult ward does a patient die from a fault in nursing, whereas in children's wards it happens too often to allow us any composure of mind. The fault is not always obvious, nor is it always in the

nurses. More often it is in the hospital itself or in the staff, who impose upon the nurses tasks beyond their capacity. The Paediatric Committee of this college in their recent report have shown where some of the faults lie; I need therefore say no more on that score, but leave it in the hope that the heavy hand of authority and precedent will be lifted by wise reform in nursing education. My purpose in discussing nursing is to draw attention to a more particular need, and one which is likely to increase in the future.

The most difficult and time-absorbing task in nursing is the care of the youngest children, who must be nursed, fed, and changed at frequent intervals of the night and day. If in addition there is much technical treatment the care of one seriously ill infant becomes one woman's work. If there must be off-duty periods, it will be two women's work. If we introduce the three-shift system, it will be three women's work. While these infants are in hospital their mothers are at home suspended in anxiety. It would seem logical, therefore, that a solution of the problem should be found in admitting the mothers to the hospital to nurse their own children. This is no theoretical proposal. I have worked under this arrangement in my hospitals for many years, and I count it an indispensable part of nursing in a children's unit. Nor is it a revolutionary idea. By far the greater part of sick children's nursing is already done by mothers in their homes, and the mother's nursing unit is merely an extension of this responsibility. Not all illnesses will be suited to this nursing, but the majority of all children under the age of 3 derive benefit from it. The mother lives in the same room with her child. She needs little or no off-duty time, because the sleep requirements of a mother fall near to zero when her own child is acutely ill. She feeds the child; she tends the child; she keeps it in its most comfortable posture, whether on its pillow or on her knee. The sister and nurse are at hand to help and to administer technical treatment to the child.

The advantages of the system are fourfold. It is an advantage to the child. It is an advantage to the mother, for to have undergone this experience and to have felt that she has been responsible for her own child's recovery establishes a relationship with her child and confidence in herself which bodes well for the future. It is an advantage to the nurses, who learn much by contact with the best of these women, not only about the handling of a child but about life itself. It is an advantage to the other children in the ward, for

whose care more nursing time is liberated. In teaching hospitals it is of further advantage to the students, who gain a practical experience of the form of nursing they will depend on in their practices and learn to recognize the anxieties and courage which bind the mothers to their children during illness: a lesson which fosters the courtesy on which the practice of medicine depends. I advocate this method of nursing, not on sentimental grounds but on the practical grounds of efficiency and necessity. Apart from all other reasons the shortage of nurses will impose this method on us in the future.

CARE OF CHILDREN IN SPECIAL HOSPITALS

About a third of Newcastle's children admitted to hospitals enter the fever hospitals. In some towns the proportion is higher. A considerable number of these patients are infants and young children, suffering from illnesses other than the acute specific fevers. Apart from diphtheria, which may justify this specialization of a hospital, there appears to be little reason for the sharp division between the fever hospitals and the acute medical sections of the children's department. A family is stricken with streptococcal infection. One boy develops a quinsy and stays at home; his brother has a sore throat with a rash and is sent to a fever hospital; and his infant sister gets streptococcal septicaemia and meningitis and is admitted to a children's hospital. An infant with infective gastro-enteritis which yields a dysentery organism goes to a fever hospital; another infant with gastro-enteritis which is equally infective but yields no dysentery bacillus remains in the children's hospital. Both types of hospital make their contribution to the care of children and the study of their diseases, but as time goes on each approximates to the other in its techniques and in the scope of its work. The corollary of this is that the staffing and nursing of both types of hospital should approximate also. The staffs of the fever hospitals should be trained as paediatricians and be specially instructed in the hospital care of children, and by contact with paediatric hospitals they should keep in touch with advances in the subject. On the other hand, the staffs of children's hospitals should know their fevers, and be *au fait* with all advances in their prevention and control.

So far I have been discussing institutions which are thoroughly familiar to us, institutions in which we live or work each day. If at

times we tend to neglect the comfort and emotional welfare of the children in these institutions by leaving that responsibility to others, we are quickly brought back to reality by contact with questioning parents. I now pass on to another type of institution where the care of children is not so readily safeguarded. This is the long-stay hospital—the orthopaedic hospital, the children's sanatorium, and the like—where patients may remain for months or years, where the medical staff who dictate the length of stay may know little about the parents of the children, where the parents *may never have the opportunity of discussing the effect of confinement on the future of the child with someone who has carefully considered that aspect of its welfare.*

If Samuel Butler had extended his *Erewhon* to include this problem no doubt he would have said that a decision to restrain and confine a person for 6 months for a civil offence would be reached only after hours or days of careful consideration of the evidence by a judge and a number of other trained people, but that a child, on whom the effects of confinement are much greater than on an adult, could be committed to hospital for far longer periods with *far less consideration of the evidence or the need.* I have experimented in the domestic care and treatment of children with active abdominal tuberculosis, of children immobilized by orthopaedic appliances, of children with chronic disease which requires frequent observation and examination; and from these experiments I am convinced that too often and too lightly is the decision made to confine children in long-stay hospitals. Sometimes the primary diagnosis is at fault. A child with coeliac disease may lie for months in bed under the impression that he has tuberculosis. Sometimes the assessment is at fault. A child with rheumatic heart disease may lie for months when he would be better gently idling about in his own home and garden. The crux of the matter is the careful ascertainment, before the child is admitted, of the clinical problem, of parental aptitude, of their domestic circumstances, and of the conditions of the hospital to which it is proposed to send the child. This ascertainment can be made only by people with considerable clinical experience and knowledge of society. As a doctor's experience increases the fewer are the children he confines to hospitals.

Some of the long-stay hospitals have attempted to set their house in order, with partial success. But a long-stay hospital can *never be completely successful in providing the things of which the*

child is deprived, whatever the educational arrangements may be, or however many their books, toys, and cinemas. These may mitigate the deprivation, but do not give the sense of personal attachment, the relationship, the companionship, which are necessary exercises for the mind of the growing child. I have had to listen to a great deal of evidence from men and women who spent much of their childhood and adolescence in these institutions. The sensitive and intelligent witnesses recalled with nightmare memories the long hours of winter evenings which pressed upon them in their adolescence, the aimlessness of their existence, the uncertainty of their future. They had their lessons each day, and raffia work and entertainments, but there was no intimacy with anyone who could explain to them the purport of their illness or encourage them with plans for the future. The fault lies in the form and arrangement of most of these long-stay hospitals. They have been conceived too much as medical institutions and arranged too much as hospital wards. It would be better if the children lived in small groups under a house-mother, and from there went to their lessons in a school, to their treatment in a sick-bay, and to their entertainment in a central hall. There would be no disadvantage in the house-mother having had a nursing training, but that in itself is not the qualification for the work she will do. Her duty is to live with her group of children and attempt to provide the things of which they have been deprived.

I must consider one other type of hospital, which will bring me full circle to Charles West again. He left obstetrics to concern himself with children's hospitals, so it is not inappropriate that a children's physician should turn for a moment to maternity hospitals, for in these places one-third of the children of the country begin their lives.

So far I have been suggesting that many of the faults in our hospitals are due to poverty-stricken ideas, which took their origin a hundred years ago, and to a formal method of construction and staffing which precedent has laid upon them. This is as true of maternity hospitals as of others. Some are now escaping from poverty and with coloured oilcloths, stainless steel, and plate glass are much gayer than they were—although I must confess I see vulgarity in much of this new gaiety—but there has been little reform of essentials.

What are these essentials? I take it that the function of a mater-

nity hospital is to deliver a woman safely of her child, and afterwards to care for them in such a manner as to ensure their health, to establish their intimate and interdependent relationship, and finally to leave the woman free from the fears of having another child. Our maternity hospitals are ensuring the safe delivery of a woman to a greater and greater degree, but are they fulfilling the other two functions?

I know maternity hospitals which are the hygienist's dream of perfection. The women lie for their 10 days in immaculate beds placed equidistantly along sterile walls. Their ward is a picture of calm repose and passive immobility. You ask what has happened to create this atmosphere of silence and subdued conversation and fail to get an answer; but the truth is that the mothers are mystified by an arrangement under which their babies have been taken away from them at the time when, at the end of 9 months' waiting, they had expected to possess them. The babies are away from them congregated in a room along the corridor beyond their earshot, out of sight but not out of mind. At regular intervals of the day they are placed on a trolley, wheeled along the corridor, and with the ringing of a bell which announces that milking-time is at hand, they are delivered by one masked woman to another masked woman at the door of the ward in which the mothers wait. Milking-time over, the babies are re-embarked for their nursery where they are solaced with sugar and water, while the mothers wait again with empty arms and quiet resignation.

This, surely, is physiologically wrong. I and many of my colleagues have the advantage of working in maternity hospitals where, throughout the puerperium night and day, mothers and babies are kept within reach of each other, where the mother may pick up her baby when she desires, where everything that is done for the child is done within sight of the mother at her bedside, and experience shows that with simple precautions not only is the danger of neonatal infection less than it otherwise would be, but breast-feeding and the relationship between mother and child are firmly and safely established in a physiologically natural manner.

Co-operation between obstetricians and paediatricians is now becoming close, but concerning this I have one piece of advice to offer. It is next to useless to enlist the services of a paediatrician in a maternity hospital only to visit occasionally and give advice on sickness. In a maternity hospital of any size there should be an

experienced paediatric physician who lives there, or visits each day regularly and punctually. It is his or her duty to prevent and treat sickness in the children, and to apply a knowledge of human biology on which the relationship between mother and child is founded. The paediatric physician will have his own technique of ward rounds, of control of infections, of treatment of disease, all of which demand a special knowledge. In smaller hospitals which cannot find such an experienced person for their staff, at least one of the obstetric officers should have a training in this branch of clinical work and human biology which fits him to supervise that aspect of child-care.

CONCLUSION

Although I may have failed to make it clear, the tenor of my discourse has been that the care of patients in hospitals is a subject which demands scientific study, and experience based on such study, and that the need for this is greatest of all in children's hospitals. A children's hospital is an instrument of medical treatment. It is a highly specialized instrument, and its arrangement must be constantly altered and adapted to meet changing needs. It is also a dangerous instrument in the hands of those who do not know how to use it.

These difficulties and dangers should be exposed in constant inquiry and discussion, and corrected by research. For this purpose some members of the profession, themselves experienced in the work of children's hospitals, must make themselves expert in the physical examination of institutions. No amount of administrative or clinical experience alone will fit them for that work.

XVI. *Hospital Beds for Children: an Estimate of Needs¹*

THERE is as yet no formula by which the need for hospital beds can be accurately calculated. Other things being equal, the need should depend only on the incidence and type of illness in the town or district concerned. But other things are never equal. The customs of the people, the houses they live in, the habits of the doctors, and the quickness of the hospital staffs in dealing with their patients vary from place to place and from time to time.

Of all institutions where the sick are treated, the greatest variability of staffing, of method, and of custom is to be seen in children's wards and in children's hospitals. Nor is it only a matter of staffing, method, and custom. For example, some hospitals open children's wards, not to meet a clinical need, but to satisfy a nurse-training scheme or some other professional demand. These and other variable factors suggest to us that we should attempt the estimate of hospital beds for sick children that we offer here.

THE INVESTIGATION

Our estimate is based on surveys that we made in 1943 and 1944, and which we repeated in 1950. The basis of the surveys was the child population in Newcastle upon Tyne in those years. From this total sample of about 50,000 children aged 0-12 years we identified each child who went to hospital or nursing-home. By personal examination of the records, the age of the child, the duration of the stay in hospital, and the nature of the illness were established; and duplicate admissions or transfer from one hospital to another were eliminated.

In our enumerations there was a possible source of error through some Newcastle children having been admitted to hospitals in other towns. *The child away from home on holiday or at a boarding-*

¹ Written in collaboration with Mary D. Taylor (who held a research grant from the Scientific and Research Committee of the Newcastle Regional Hospital Board). Published in the *Lancet*, 267, 1954, p. 719.

school may have taken suddenly ill, or parents may have wished their child to be treated in another city. After examination of this possible source of error we conclude that its effects are so small that it does not materially affect our estimate.

The investigation took into account all children resident in the city under the age of 13 years, but in 1950 we extended it to include the children aged 13 years and 14 years. Ten hospitals and four nursing-homes were involved in one or both surveys, some of which were situated outside the city.

The standard of record-keeping in the hospitals was high, but their methods of classification of diseases were not sufficiently informative for our purpose; so we designed and used a classification and record which indicates more clearly than the traditional classification the type and scope of the work undertaken by the hospitals. We consulted the information collected by the Regional Hospital Board for the Ministry of Health on their S.H. 3 return, but this was of no value to us.

LOCAL FACTORS

In interpreting the results we would ask the reader to keep in mind certain factors favouring an economical use of beds in Newcastle, which might not obtain in some other places.

1. The hospital accommodation for children was ample, and there was no waiting-list for admission, except for tonsillectomy and operation for squint.

2. The threshold of admission to hospital was high, because of the prevailing hospital policy that whenever possible the sick child should be treated at home.

3. This policy was reinforced by the provision of a daily out-patient consultation service to which the family doctors could refer their patients immediately. Conducted by senior and experienced consultants, this service reduced the demand for beds.

4. The children were admitted mainly to children's hospitals or children's departments arranged and staffed as combined clinical units, in which personal consultations between paediatric physicians, surgeons, and specialists were so promptly obtained that time and anxiety were saved.

5. In these children's hospitals and departments all children were examined by an experienced paediatric resident at the time

of admission to hospital, and the allocation of patients in wards and beds was under his control with consultant advice immediately available. *These measures eliminated outbreaks of those infective illnesses for which children's wards are often closed, and in other ways contributed to a full and efficient use of bed accommodation throughout the periods of survey.*

6. In two hospitals time was saved by using, for the nursing of sick children, the Babies' Hospital method of 'mother-nursing' *which shortens the child's length of stay in hospital.*

7. Newcastle being a regional and university city, its hospitals are self-sufficient in their consultant and specialist services. *Therefore the estimates we make are for similar cities. For those towns and districts which are not self-sufficient in paediatric and specialist services, and from which transfer of cases to a regional centre will take place, a reduction in the estimate is required.*

8. If our estimates are to be used for policy and plans in the future, the changing trends of mortality must be noted. The disappearance of diphtheria (185 admitted in 1943, and 0 in 1950), the diminished hospital needs for scarlet fever (495 in 1943 and 26 in 1950), are on the credit side. On the debit side is the long stay in hospital for modern forms of treatment of children with tuberculous meningitis; but it can be predicted that after about 10 years there will be a steady reduction in the number of children coming to hospital for treatment of tuberculosis. On the debit side also is the increase in tonsillectomy, but it is within the bounds of possibility that a changing attitude towards this operation will diminish greatly the demand that it shall be done.

9. A local policy about scarlet fever exercises a paramount influence on the need for hospital beds. In Newcastle the policy, which we hold to be the right policy, is to admit to hospital only those children with scarlet fever, the severity of whose illness indicates the need for hospital treatment. In some other towns all cases of scarlet fever are admitted to hospital, however mild the illness may be.

THE POPULATION

Estimates can be based on (a) the total population (Table 24), or (b) the population of the age-groups under study (Table 24), or (c) the birth-rate (Table 25). Our final estimates are based on the

first of these and are expressed as rate per 100,000 of total population. This is probably the best method of comparing one town with another, but for strict comparison the governing factor of birth-rates should also be considered.

TABLE 24
Population of Newcastle upon Tyne

	<i>Total</i>	<i>Children</i>
1943	254,890	49,800 under age of 13
1944	262,920	50,400 " " 13
1950	294,800	57,800 " " 13
		66,130 " " 15

TABLE 25
Annual births and birth-rate

	<i>Live annual births</i>	<i>Newcastle birth-rate</i>	<i>National birth-rate</i>
1943	4,548	17.8	16.5
1944	5,359	20.4	17.6
1950	5,051	17.14	15.8

THE FINDINGS

There is so little variation between the years of 1943 (3,782 admissions) and 1944 (3,704 admissions) that the figures for these 2 years are dealt with as an annual average of 3,743 children (aged 0-12 years) admitted to hospital in each year, which is 7.48 per cent. of the childhood population in that age-group, or 1,410 per 100,000 of the total population. In 1950, 3,980 children aged 0-12 years were admitted, which is 6.85 per cent. of children in that age-group, or 1,350 per 100,000 of the total population. In 1950 there were in addition 260 children aged 13 or 14 admitted to hospitals. These figures show that 1 child in 15 is admitted to hospital each year, which is equivalent to every child being admitted to hospital once before the age of 15.

The admissions for tonsillectomy and scarlet fever play so large a part in the figures that they must be considered separately. In the years 1943 and 1944 the average annual admission for tonsillectomy was 890, and in 1950 it was 1,482. For scarlet fever the numbers were 495 in 1943 and 1944, and 26 in 1950 (Table 26).

TABLE 26

Annual admissions in age-group 0-12 years

	<i>Total</i>	<i>For tonsillectomy</i>	<i>For scarlet fever</i>
1943 }	3,743	890	495
1944 }			
1950	3,980	1,482	26

An analysis of admissions is given in Tables 27 and 28, and we recommend that annual reports of children in hospital should be presented in this way and extended so that each category of illness is related to age-groups, to length of stay, and to results. This information is necessary if the type of clinical work in hospitals is to be judged.

TABLE 27

Annual admissions in age-groups

	<i>Infants (0-12 mths.)</i>	<i>1-4 years</i>	<i>5-12 years</i>	<i>13-14 years</i>	<i>Total</i>
1943 }	419	1,293	2,031	(not counted)	3,742
1944 }					
1950	449	1,197	2,334	260	4,240

The ages of children (Table 27) are grouped in these three categories: (1) infants (aged 0-12 months); (2) pre-school children (aged 1-4 years); and (3) school-children (aged 5-12 or 5-14 years).

Our estimates of the lengths of stay, which we use later, were made from the following groups: (1) for a period of less than a month; (2) for 1 to 2 months; (3) for 2 to 3 months; and (4) for 3 months or more.

CALCULATION OF BED REQUIREMENTS

For tonsillectomy

In 1943 and 1944 there were 890 annual admissions of children to hospital for tonsillectomy. In 1950 there were 1,482 up to the age of 12, and 1,574 up to the age of 14. The 1950 figure represents a higher-than-normal level when waiting-lists were being worked off. As an estimate for the future we will take a figure of 1,200

TABLE 28

Annual admissions to 'acute' hospitals for chief categories of disease

	1943-4 (0-12 yrs.)	1950 (0-12 yrs.)	1950 (13-14 yrs.)
Scarlet fever	495	26	0
Diphtheria	185	0	0
Measles, pertussis, &c.	143	62	0
Poliomyelitis	10	65	1
Meningococcal infections	34	27	0
Upper respiratory infection (including tonsillitis and otitis media)	140	364	28
Lower respiratory infection (including pneumonia)	230	161	0
Acute gastro-intestinal conditions (in- cluding dysentery)	213	126	0
Acute abdominal surgery:			
Appendicitis	42	67	14
Intussusception	13	28	0
Pyloric stenosis	15	21	0
Others	8	8	2
Trauma:			
Burns	55	33	0
Head injuries	56	49	3
Fractures	76	80	8
Poisons	7	8	0
Minor injuries	114	65	0
Planned surgery:			
Tonsillectomy	890	1,482	92
Eye operations	40	114	15
Plastic operations	12	32	0
Other planned surgery (including ortho- paedic)	127	232	9
Sepsis	140	70	5
Osteomyelitis (or suspected)	19	28	0
Tuberculosis:			
Meningitis or miliary	26	30	1
Bone and joint	11	10	0
Others	90	37	3
Other conditions:			
Acute rheumatism or chorea	24	21	4
Nephritis	11	16	0
Venereal diseases	7	3	1
Miscellaneous skins (including im- petigo)	110	17	2
Bronchiectasis, asthma, and empyema	17	28	1
'Feeding problems'	25	14	0
Other alimentary disorders	50	74	
Acute unknown infections	33	36	

tonsillectomies a year in children up to the age of 12 years, and 1,250 up to the age of 14 years. This represents 1·8 per cent. each year of the child population at risk, and 24·7 per cent. of the annual births. Assuming that each child spends either 2 or 3 days in hospital and that each bed used for this purpose accommodated 2 patients a week, 12 hospital beds would suffice for these 1,200-1,250 children admitted for tonsillectomy—i.e. 4 beds per 100,000 of the population.

Length of stay

A distinction must first be made between the admissions to 'acute' hospitals and the admission to 'long-stay' hospitals, or children's sanatoria; but the latter form so small a part of the whole (30 admissions in 1943 and 88 in 1950) that we will confine our estimate of long-stay beds for children to the statement that about 35-45 will be required for a population of 300,000, or 12-15 per 100,000 of the population, assuming that the average stay in that kind of hospital is 6 months; but this estimate will vary according to the clinical policy of the hospitals, and its adjustment to the wishes and capabilities of the parents. This estimate of long-stay beds may be on the high side.

For all other categories of illness (i.e. excluding tonsillectomy and long-stay illnesses in special hospitals, for which we have already made a length-of-stay estimate) we have calculated an average length of stay expressed as the number of children per bed per year, by analysing the records of three fully staffed and active children's departments or hospitals, which admit about 4,000 children a year with all types of medical, surgical, and special illnesses. A few of them (40 in the year) were in the wards for more than 3 months, and 150 for 1 to 3 months, but these were included in our calculation of the 'average length of stay'.

The average length of stay (excluding those in hospital for tonsillectomy) was just over 14 days. From this we estimate that each hospital bed would accommodate 23 patients a year, making due allowance for turn-over and other adjustments.

In 1950 for the Newcastle children in the age-group 0-12 years there were 2,410 admissions for illnesses in this category of medical, surgical, and special diseases. According to our estimate, 105 beds would be required for these. Adding the group of children aged 13-14 years, 110 beds would be required.

Final estimate

Assuming that existing conditions continue, Newcastle, with its population of 295,000, including 66,130 children in the age-group 0-14 years, will require—for all types of illness other than those admitted to sanatoria or similar long-stay hospitals—the following number of hospital beds:

For tonsillectomy . . .	12
For other illnesses . . .	110
Total . . .	122

In other terms this is 42 beds for children aged 0-14 years, per 100,000 of the total population. In addition we have tentatively suggested that 12-15 beds per 100,000 of the population would be required for long-stay cases in special hospitals.

FINAL COMMENT

This estimate of 42 children's hospital beds per 100,000 of the population is for an industrial town in the north of England with a high birth-rate and a high morbidity incidence. It is a tight estimate: to be on the safe side, the figure should be set at 50 per 100,000. On the other hand, fewer beds would be needed in some other towns, and considerably fewer in those districts or towns which are not self-sufficient in specialist and paediatric staffs.

This number of beds suffices for all hospital treatment of children, other than for what goes on in sanatoria and maternity hospitals.

In a university town which is a teaching and specialist centre still more beds will be required to meet regional needs.

This estimate may serve as a guide to the use of beds. We have found that hospital beds for children are used more effectively—i.e. with a higher turnover in numbers—and with greater satisfaction to patients, to parents, and to staff, if the children's wards are grouped and arranged together in children's departments or hospitals, of convenient size, which bring the medical, surgical, and specialist staff into a close and unavoidable working relationship. This way works better than scattering children's beds in specialist departments.

While our estimate may serve as a numerical guide, it cannot be used to judge the quality or value of clinical work, or the relationship of staffs and the arrangement of their responsibilities, on which so much depends in children's wards. The worth of these is measured, not by time-motion studies or by enumeration, but by the judgement of those who are fit to judge.

PART V

*The Practice of Medicine and
the Training of Doctors*



XVII. *Paediatrics in Universities and Teaching Hospitals*¹

FOR want of a better word, paediatrics will here be used to denote that branch of medicine which is concerned with preserving or restoring the health of children. Its functions are to understand the principles of growth, health, and disease in childhood, and to apply methods of preventing and curing the diseases of children. It might best be called 'child medicine'.

It is the purpose of this memorandum to examine the present state of paediatrics in this country, to consider the part which universities and teaching hospitals should take in promoting the subject; and to describe the function and the organization of Departments of Paediatrics in universities and teaching hospitals.

It is necessary first to consider if it is right to encourage the development of paediatrics as a separate subject. There are those who foresee a danger in this. They plead that such a development might ultimately separate the medicine of childhood from the medicine of adult life, and that this would not be in the best interest of medical practice. Admittedly a complete division of the practice of medicine in that way would be harmful and dangerous, but there is no need to fear that the development of Paediatric Departments would bring about that result. *Indeed, if they were organized for research and to give an adequate training in paediatrics to students, practitioners, physicians, and specialists they would have the opposite effect. They would give to the practitioners of medicine and surgery a clinical experience which most of them at present lack, and an understanding of the nature of disease in infancy and childhood which would clarify many of the problems of disease in adult life. Beyond this the fact remains that the profession is at present spending most of its time and energy in studying and treating the diseases of senescence and of old age, while by comparison the prevention and cure of disease in childhood remains*

¹ A Memorandum for the Trustees of the Nuffield Provincial Hospitals Trust. Published by the North-Eastern Regional Hospitals Advisory Council, 1942.

neglected. Moreover, it must be realized that already many doctors have entered a career of clinical paediatrics in public health and hospital services, and for the training of these alone if for no others the universities should establish departments of teaching and research.

A final argument for the development of paediatrics is the need for research work. This is particularly necessary if we are to advance our understanding of the nature of disease in early infancy of which it is not too much to say that there is no field of clinical research which is so neglected but which is likely to yield such fruitful results.

The present state of practice in paediatrics is best surveyed by examining the fields in which it works. These are:

1. General practice.
2. The public child health services.
3. Consultant and specialist practice.
4. Hospitals and other institutions for the treatment of children.

PAEDIATRICS IN GENERAL PRACTICE

The treatment of sick children forms about one-third of the work of general practice. This proportion has fallen where the Child Welfare Centres have taken over the work of advising parents on the hygiene, feeding, and minor ailments of their children under the age of 5. But it is to the general practitioner that a child is taken when seriously ill, and therefore on him falls the prime responsibility of diagnosing and dealing with disease. Considering how little experience of diseases of children medical practitioners are able to obtain either as students or as hospital residents, the majority of them must be given credit for the standard of paediatric work they attain in practice. This is not to say that their diagnostic and curative skill is satisfactory or could not be improved, a fact which practitioners themselves are the first to admit.

The influence of the Child Welfare Service Clinics on medical practice deserves notice. Many of these clinics are staffed by whole-time medical officers who have little or no contact with the local practitioners. Their time is taken up in a circuit of clinics in which they advise on matters which an intelligent mother or nurse already understands. On the other hand, the family doctors often have no opportunity of seeing their child patients until and unless they are

seriously or acutely ill. This divorce between the work of the clinics and the work of medical practice has caused the general practitioner to relax his interest in the welfare of the healthy child. It would not be difficult to alter and reform this, but a first step must be a recognition of the faults both in the clinic system and in the clinical practice of paediatrics, and this must be followed by a well-conceived scheme of post-graduate education, with the object of spreading a knowledge of children's diseases to clinic doctors and to medical practitioners, and of arousing an interest in hygiene, feeding, and other factors which promote health.

PUBLIC CHILD HEALTH SERVICES

The chief of these are the Child Welfare Service and the School Medical Service.

There are in this country 3,200 child welfare centres of which 2,400 belong to local authorities and 800 to voluntary associations. We can estimate that at least 1,200 medical practitioners are working in these centres advising on hygiene, health, and minor ailments. Some are whole-time medical officers, engaged only in this type of work or combining it with maternity work in ante-natal clinics. Local authorities spend £3,100,000 a year on their maternity and child welfare services. Including the voluntary associations about £3,000,000 a year must be spent on child welfare centre work alone. The cost of 'school medical services in association with elementary education' for 1932-3 (later figures not available) was £4,300,000 a year. Of this £2,600,000 was spent on medical inspection and treatment. It is safe to estimate that this latter figure is now well over £3,000,000.

Considering the large amount of money spent on these services and the influence they have, or might have, on child health, they receive very little assistance from those who are primarily responsible for the advancement of medical science. University Paediatric Departments might be of great assistance to those services. Some members of the teaching Paediatric Department could act as consultants and advisers to them, and their hospitals and clinics should serve them as consultation centres. To support this view we can look at such towns as Toronto, Chicago, Gothenburg, and Leyden, where some of the greatest achievements in promoting child health have been attained, through the influence on local

health policy of paediatric physicians engaged in teaching and research.

A matter which closely concerns teaching Paediatric Departments is the lack of special preliminary training, or later post-graduate training, for those who undertake a whole-time career in these public child services. Less than 10 per cent. of the staffs of the child welfare and school medical services have held resident appointments in children's hospitals. Though the services themselves have often not seen the necessity of instituting this training for their doctors, the universities who have failed to provide the training are at least equally to blame. Something must be done to correct this, and its correction will throw a considerable responsibility on teaching hospitals and university Paediatric Departments.

CONSULTANT PAEDIATRIC PRACTICE

Four things are necessary to make a consulting physician or surgeon *competent in his work and to gain for him the recognition* of other doctors who will desire to draw on his experience and seek his advice. The *first* is a special training in his subject; the *second* is a continuing experience in his special work; the *third* is a close contact with colleagues and fellow workers; the *fourth* is that he shall not be distracted by economic or other causes from his work.

In the past it has been difficult for a sufficient number of paediatric consulting physicians to fulfil these four requirements, and the result has been that there is now a totally inadequate number of trained consulting paediatric physicians in the country. The cause is mainly an economic one. Most consulting physicians are also teachers in the medical schools, but receive little or no payment for this. (The seven provincial universities spend only a *total* of £410 a year on paediatric teaching and research, and the London teaching hospitals not much more.) They have had to rely therefore on practice for a living in order to devote themselves to teaching and research. The amount which can be earned in paediatric consulting practice is small and therein lies the chief difficulty in attracting a sufficient number of doctors to take up this branch of work. The leading paediatrician in a big provincial teaching town often earns less than £1,000 a year from consulting work in paediatrics, while other trained paediatricians under the age of 35 earn no more than £300 or £400 a year in that way. This represents a net

income of from £200 to £650. They must therefore supplement their income from other sources, which becomes a distraction. The economics of consulting paediatric practice is illustrated from a pay-bed department of a teaching hospital. In one year the total of medical and surgical fees paid was £9,600. Of this amount the fees paid for the treatment of children was £325. Of this £325 only £34 was paid to paediatric physicians. Another illustration is seen in figures which came to light during an investigation into the infant deaths which took place in a large city during 1939. Only one of these had been seen in private consultation by a paediatric consulting physician before its death.

These economic problems of consulting practice do not concern the universities, but since the staffs of Paediatric Departments in universities and teaching hospitals must in future consist in part of consulting paediatricians, the difficulty there is in attracting a sufficient number of men and women of high ability to take up a career in paediatric teaching and consulting work should be recognized.

HOSPITALS AND OTHER INSTITUTIONS FOR THE TREATMENT OF CHILDREN

The story of the hospital and institutional treatment of children is one which has not been revealed to the public and is too little realized by the medical profession. The true facts have been obscured by false sentiment. They are that unless a children's hospital ward is administered in the right manner, nursed by the right nurses, and staffed and supervised by the right doctors, they will do more harm than good. That some hospitals and other institutions have in the past done more harm than good to children is beyond dispute. There are two main dangers. The first is the physical harm and death which come to infants and young children from hospital infections. The second is the psychological harm which may come both to younger and older children who are emotionally and mentally starved from too long a stay in these places. To correct and control this imposes a heavy duty on the medical staffs of children's hospitals. They must concern themselves with the details of the welfare of their patients to a greater degree than is necessary in adult hospitals. They must attend their wards more frequently and constantly than in an adult hospital. New ideas

about the arrangement of children's departments and children's hospitals are rapidly arising. The rejection of the old division of medical and surgical wards; the rearrangement of the wards according to the age-groups of the children; the admission of mothers to undertake the nursing of the infants; the separation of the 'short stay' patients from the 'long stay' patients; the provision of instruction and education whenever possible—these and other innovations will alter children's hospitals. Some of the teaching children's hospitals have already been leaders in bringing about these changes. But much more remains to be done before the institutional treatment of children can be considered satisfactory throughout the country, and here again the university paediatric hospitals will have to play a part in setting a standard of hospital arrangement and treatment.

PAEDIATRICS IN UNIVERSITIES AND TEACHING HOSPITALS

In the preceding paragraphs it has been suggested that reforms and improvements in the paediatric services are not likely to be effective unless they are based on a better training of medical personnel, and on development of research into the problems of health and disease in childhood. It can be conceived that research and teaching institutions outside the universities and medical schools might arise to undertake these duties, but if that were to happen and thereby bring about a decline in the responsibility of the universities and teaching hospitals towards the subject of paediatrics, the ultimate results would be harmful to medical education. Assuming that the universities and teaching hospitals will recognize their responsibility, they should be advised on the part they should play.

At present in the English medical schools the teaching of paediatrics varies in the different medical schools from a short course of lectures and clinical demonstrations to a 2 or 3 months' course of intensive training and clinical work. There is only one Chair of Paediatrics in England, and none in London. The British Post Graduate School of Medicine in London has no properly constituted Department of Paediatrics. Figures from the Reports of the University Grants Committee show that the provincial universities in England spend only a total of £400 a year on paediatric

training and research, varying from £20 a year in Sheffield to £90 a year in Bristol. London University through its twelve Medical Schools spends only £1,400 a year on paediatrics. In Scotland there are two complete and well-established Departments of Paediatrics at Edinburgh and Glasgow, each of which receives from the university about £2,500 a year. Fortunately, the amount which universities spend in this country is no index of the position of English paediatrics, the reputation of which stands high in some centres through the initiative and enthusiasm of a few individual teachers. An outstanding example is Birmingham where there is a Chair of Paediatrics, and the Children's Hospital forms an active centre of research although the university's contribution is only £56 a year.

If it be conceded that paediatrics is no minor speciality but a major part of medicine itself, that its development is necessary both for the science and practice of medicine, and that it is an instrument of great value in the instruction and training of the medical student, then the part which universities should play becomes clear. Each university should establish a Department of Paediatrics adequately staffed for teaching and research based on a children's hospital or children's department fitted for the work. They should seek to do this in a way which fosters a close contact between paediatrics and other branches of medicine, and with the University Departments of Pathology, Anatomy, and Physiology. It would be valuable also to foster a collaboration between paediatrics, which concerns itself closely with the whole welfare of the child, and the non-medical social sciences which are also concerned with the child.

Until the universities take the step of establishing these departments, we shall not be able to attract young doctors of high ability to this branch of medical work. Under prevailing conditions, we are turning out in each year from the medical schools of this country less than ten paediatric physicians who can be considered to have been adequately trained in suitable post-graduate appointments, and therefore fit to hold teaching appointments in university departments, or higher administrative posts which require a knowledge of paediatrics.

While the function of a university paediatric hospital department is clear, its form will vary according to the available resources and staff. It must be based on a hospital with sufficient clinical material,

and it would be best placed in a children's hospital with not less than 100 beds associated in a hospital centre with other teaching departments. It will be easier to achieve this in the provinces than in London.

The functions of the department will be to provide:

1. A course of training in children's diseases and child health for medical students.
2. Post-graduate teaching for medical practitioners and the medical staffs of the child welfare and school medical services.
3. An advanced training through clinical and research appointments for those who wish to pursue a career in paediatrics.
4. Opportunities and facilities for research.

The medical staff should consist of:

1. The head of the department who will be a paediatric physician devoting all or the major part of his time to teaching, organization, and to research.
2. A whole-time assistant with the status of university lecturer.
3. A senior staff of 5 or 6 physicians or surgeons who will have been trained in paediatrics, and some of whom will practise as consultants in paediatrics or general medicine or surgery.
4. An associated visiting staff of specialists in otology, ophthalmology, child welfare, medical genetics, and other subjects.
5. An associated non-clinical staff, including particularly a pathologist, who should, if possible, be chosen for the work by the University Professor of Pathology and be a member of his department.
6. A junior whole-time staff of research assistants and registrars whose appointments would last 3 years.

The estimated departmental budget for salaries and research expenses which the university should pay is about £4,000 a year.

XVIII. *The Need for Understanding the Individual as Part of the Training and Function of Doctors and Nurses*¹

I DETECT an anxiety in the minds of those who have chosen this subject for discussion, as if medicine and schools were wilting through lack of understanding of the individual or have gone too far in techniques at the expense of human care. Fully to understand the nature of our fellow men may be beyond the reach of most of us, life being short and the art too long for that. But for the ordinary affairs of life it is easy enough to understand the individual. The difficulty is to know what to do with him or to bring ourselves to do it. Nevertheless, for the purpose of this discussion, we can accept three premisses—that doctors and nurses, because of the intimate character of their professional work, need particularly to understand the individual and to consider his feelings; that some are better than others at this; and that we should do something about it if we can.

There appear to me to be three kinds of understanding of individuals. There is the basic understanding which comes by the process of trial and error in the experience of close human relationships, and finds expression in proverbs and precepts. This is the currency of our daily lives and is measured in terms of harmonious living with family and friends. Secondly, there is the expert understanding of abnormal types which comes by particular study and trained observation. Thirdly, there is the specialized understanding of one or other aspect of the individual, which is seen, for example, in the industrialist who selects with unfailing skill a man for a particular job, by understanding that quality for which he employs the man. This specialized understanding usually operates successfully only within its own limits. Outside these limits it may go quickly astray, as is seen in the example of this same skilled industrialist, who readily falls prey to any crank or charlatan when he chooses a doctor to tend his body or an artist to paint his

¹ Delivered at a Conference on Mental Health held in March 1949. Published by the National Association for Mental Health, 1949.

portrait. There are many people with the specialized understanding who show little basic understanding of individuals outside their specialized activity.

The capacity to understand the individual in all of these three ways depends on our sensitivity, our intelligence, our experience, and our training. But not on these only. To understand we must see clearly, and we do not see clearly if we are blinded by envy, pride, personal ambition, jealousy, fear, or greed. No amount of intelligence or training will make us understand if our observations are deflected by these distorting influences. So to end this introduction, I should say that to understand the individual the doctor and the nurse must gather their experience and train their powers of observation in a spirit of sympathy, charity, and magnanimity, and that this capacity is therefore on another and perhaps higher plane than mere scientific observation.

Our understanding of the individual begins in the apprenticeship to life which is childhood. This early experience is the richer if it is gathered in the companionship of other children of different types and different ages, in the streets, in the hedgerows, or on the village green, free from the restriction of adult supervision. We may then be too young to foresee amongst our companions any 'village Hampdens', or 'mute, inglorious Miltons', but we can begin to learn by withstanding the little tyrants of the fields, by sharing loyalties, by enduring exploits, by proffering help, by accepting aid. If some of us failed to get this experience in childhood it may be because we lived too much in a life arranged for us by adults. I am not here pleading for the go-as-you-please type of education, but the child must have its freedoms and its solitudes if its experience is to be transmuted into an understanding of individuals. Nevertheless, this transmutation will be the better if the experience is gathered against the background of a wise parental discipline and a family code of morals. Manners maketh man. It maketh also for the understanding of man.

This apprenticeship to understanding, 'plucked from the hedgerow of experience', as Mr. Ernest Bevin once described it, goes on through adolescence, but even on the verge of manhood it is still incomplete. How incomplete is revealed in the common experience of war. There we saw men, whom, in the intimate life of the university, we had mistaken for giants amongst us, and were now exposed as very weak brethren, while the quiet and the meek stood

out in unsuspected nobility. It has been said that to understand an individual you must see him in love, in cards, and in drink. Better still to see him also in the stress and strain of battle or of domestic hardship. Doctors and nurses are privileged to come near to those occasions.

I make this preliminary arrangement of my subject with some diffidence. For although my professional life has been motivated by an interest in individuals, so that any train journey is an adventure to me, and although I have followed the Aristotelian precept of judging the nature of a thing through its full development by watching a generation of children grow to maturity, trying thereby to deduce effects from causes, I remain chary of generalizations. So my heart warmed to the man on the Brains Trust who, after his colleagues had let forth a column of exhortatory advice in sentences beginning with 'of course', refused to engage himself, with the excuse that he was not one who presumed to tell other people how to arrange their lives. He spoke like an understanding man.

Does a doctor need to understand the individual more than the lawyer, the teacher, the industrialist, or the administrator? If life were merely a matter of economics and jurisprudence we would not make this claim. In those circumstances it would be sufficient to cultivate only the specialized understanding of function, the recognition of the type of man who fits a particular type of job and the extent of his failure. But, fortunately, the good life is not a matter of economics. It is a matter of manners, of ethics, and of moral philosophy, with charity as its safest guide for most of us. Looked at in this light, the doctor needs to know the individual with basic, expert, and specialized understanding if he is to work with success. He sees men of all ages from childhood to senility. He is present at birth and at death. He observes man in his confidence of full health and in his fear of sickness. He observes him near the noon of day when courage is at its height, and in the small hours of morning when it so often ebbs away. Not only must he understand the individual but he must understand him in many of these variations from the norm.

The real work of a doctor is only faintly realized by many lay people. It is not an affair of health centres, or public clinics, or operating theatres, or laboratories, or hospital beds. These techniques have their place in medicine, but they are not medicine. The essential unit of medical practice is the occasion when, in the

intimacy of the consulting room or sick room, a person who is ill, or believes himself to be ill, seeks the advice of a doctor whom he trusts. This is a consultation and all else in the practice of medicine derives from it. The purpose of a consultation is that the doctor, having gathered his evidence, shall give explanation and advice. The explanation may be that the patient has appendicitis, and the advice may be that he should go to such and such a hospital to be operated on, by such and such a surgeon. But the purpose of the consultation is not the diagnosis or the technical treatment. It is the explanation and advice, with the diagnosis acting as a means to those ends. The explanation is pursued further with what the Greeks called *pronoia*, which is something more than prognosis. With this the doctor uses his understanding of the individual and of his disease to prepare him and his near relatives for the hospital, for the technical treatment in the hospital, and for his life after hospital. It is not difficult to realize something of the intimacy, the courtesy, and the understanding which is required in this work. It takes place in the sick room of a house or in a consulting room, but sometimes even on the doorstep. In a proper consultation no third person should be present unless it is the parents or near relatives of the patient. You will see how easy it is to go astray from this conception of medicine in our design of hospitals and clinics.

If it be the purpose of medicine to give explanation and advice in consultation as a prerequisite of technical treatment, how are we to train our students? How are we to give them the understanding of the individual they will need in consultation? How are we to maintain in doctors the sympathetic understanding of so many individuals, without which their work becomes a weariness of spirit and flesh? Can we in fact teach the art of consultation?

We can excuse those who do not like this way of medicine. They are more interested in things than in people, and they will find their place by working in laboratories or as technical therapists. Concerning the others who must practise in this way, it surprises me how many of them pick up the art of sympathetic and wise consultation without any formal training in it. Perhaps they have come under the influence, even for a brief time, of a wise practitioner of medicine who gives them a pattern in his example. Perhaps it goes farther back than that, and is the fruit of inborn sensitivity and good manners learned in childhood. But in the training of our doctors and nurses we must act in the belief that to

understand the individual is an art which can be cultivated, so it is worth our while to demonstrate it.

Medical and nursing education is at present restricted to medical schools, training schools, and hospitals. Within these limitations, the art of consultation, by which understanding cometh, is best demonstrated in the rooms of out-patient departments. It is not easy, in these places, to arrange a form of out-patient consultation in which the students can share. We do what we can in our department to demonstrate its method and purpose, hoping that the student and nurse will learn from example, but they can get no more than a glimpse of the art and skill, because of the paradox that in most cases it is impossible to conduct a satisfactory consultation in the presence of a third person. We try to set aside the out-patient experience solely for the purpose of demonstrating this art and technique of consultations to the students, and feel justified in doing this, because they have already learned their techniques of diagnosis and technical treatment in lectures, laboratories, wards, and other places. The consultations are given by the most experienced members of the staff. Two or three students are allowed to watch and listen with as much self-effacement as possible. They observe the greeting. They hear the questioning. They watch the examination, and, finally, they listen to the explanation and advice. They do not examine the patients. When the students have accepted this role they appear to appreciate the occasion. They share in the sense of relief when the explanation removes brooding fears. They may even feel a little of the anguish when a tragedy unfolds itself. After the patient has left the room, discussion centres on the consultation, while the pathology and diagnosis of the disease is commented on only briefly. The choice of words used in explanation is reviewed. The restraint of words in giving advice is recalled. An experienced teacher using this instrument can thereby reveal to a student a great deal about individuals and about life itself, which cannot otherwise be seen in hospitals. I have mentioned the paradox of being unable satisfactorily to demonstrate the art of consultation in the presence of a third person. The students are made aware of this in a preliminary talk. They are told that before explanation and advice can be given to a patient they must make three diagnoses—the diagnosis of the disease, the diagnosis of the concept or fears of the disease in the mind of patient or parents, and, thirdly, the diagnosis of the patient's

capacity to understand the explanation and follow the advice. They are told also that it is the patient's privilege to be interviewed without students being present if they so desire it. Less than 10 per cent. demand this privilege, and for them it is readily provided.

With this preliminary explanation students are quick to see the significance of consultation, and some of them confess that through this experience they realize for the first time what the practice of medicine means. Lest their hopes fly too high, we bring them to earth with the reminder that between these vivid episodes there will be a great deal of dull certificate writing, for of such is the practice of medicine. We encourage them also with the advice that they will understand the trials of practice better with a touch of the Meredithian concept of human nature than by an acceptance of the tragic Shakespearian view. That is pedagogic trickery, but it keeps them alert.

A few examples may illustrate consultation and *pronoia*. It happens that a woman seeks advice about her mentally defective baby. She herself says nothing about its feebleness of mind. She may know it or suspect it, but she talks of other things. With *pronoia*, or the knowing of things about a patient without being told them, the doctor estimates the woman's notion of the situation, her attitude towards it, and her capacity for dealing with it. This enables him to choose the right questions with the right words. The mother's answer will tell him what he wishes to know without raising unnecessary hopes or fears. According to the age of the child, the attitude of the parents, and their state of mind, he frames his explanation and advice. So far the consultation goes. But beneath this encounter there is much which is not told that the doctor is aware of. The woman probably carries a deep sense of guilt that the child's condition may be due to something she has done or left undone during her pregnancy, such, for example, as the use of abortifacients. But she fears to confess it, not daring to face the crucial question. The doctor knows this without being told. So he takes care to remove this corroding guilt by saying in the course of his explanation, that the child's feebleness is due to no fault of hers, or to anything she may have done during pregnancy. He may not be able to cure the child, but by understanding and *pronoia* he can cure the mother's morbid sense of guilt.

Another example will show the lack or lapse of understanding. I was consulted by a woman with her child whom she feared might

have tuberculosis. Her fear, which she did not at first confess, was based on the resemblance of her child's ailment to that of a cousin who had sickened and died of tuberculous meningitis a few years before. The child had a transient and harmless ailment for which a quick and prompt recovery was foretold. All this was explained, and advice was given that a skin test should be done to confirm the diagnosis. For this I turned to the nurse and said, 'one in a thousand, please nurse', wishing her to bring to me a 1 in 1,000 dilution of test fluid. I was too obtuse or otherwise engaged to notice the alarm that must have come to the mother's face when I said these words, but it was reported to me later by someone who visited the woman in her home and found her stricken with anxiety and resentment. It emerged that she had mistaken my words and thought I said, 'A one in a thousand disease, nurse'. This was the source of her distrust, that I could with one voice assure her that her child was not ill, and with another tell the nurse that we had a rare or 'one in a thousand' disease. These facts were revealed only by chance. How often must it happen that ill-chosen words do harm that we know not of?

If the hospital fails to provide the medical student or young doctor with the expert and specialized understanding of the individual patients, they may gain it for themselves afterwards in the responsibility of practice. But what should be said of the nurse whose whole professional life may be spent in an institution with little time for intimacy with her patients, and her duties increasingly directed to doing things to her patients rather than for them?

Our system of nurses' education may attempt to introduce them to the need for understanding the individual patient, but being an art it is better learned by example and exercise than by precept and lectures. In pursuit of this art, the most valued lessons will come from observing their ward sisters and the doctors. The ultimate responsibility for the comfort and nursing of the sick is, or should be, in the hands of the ward sister. In this the doctors are responsible to her and not she to the doctors. With the acceptance of this authority and responsibility the ward sister will turn her attention to ways and means of educating her nurses in the courtesy, sympathy, and understanding required in the nursing of the sick.

I will mention a few practical exercises which a ward sister might follow in order to demonstrate the need for understanding the individual patient and his feelings. The first is to change from

time to time the position of the patients in the ward for the better arrangement of companionship, and not only for the ease of technical treatment. Frequently to do this in the presence of young nurses is a good way to demonstrate the need for this understanding. The second is a simple exercise. It is to learn to enter and leave a room quietly. This is better demonstrated in an out-patient consultation service than in wards with their wide-open spaces. The third exercise is the smile of greeting, one of nature's most elementary instruments to open the way to understanding. I would not go so far as to suggest the arrangement of 'charm classes' in our preliminary training schools. It would be sufficient to remember that a nurse's sour look may be as harmful as a septic finger, and if she cannot overcome it, it were better not to be a nurse. The fourth is the choice of conversation, but this is almost beyond the wit of man or woman consciously to demonstrate. To be interested rather than interesting is the first rule of conversation. This might be told to the nurses, and the descending scales of a patient's interest might be studied. The normal man's primary interest is in himself, next in his family, next in his immediate surroundings, next in his work, next in his hobbies. The normal woman's interests are of a slightly different order. Conversation directed to these interests may be flavoured with a little cajoling, teasing, sympathizing, or serious attention as befits the occasion. But as it is a chastening experience for any broadcaster to have his recorded voice played back at him, so it would be equally corrective for a nurse to have her small talk recorded for her hearing. It might then subdue the bright young thing who is constantly cheerful in brittle talk, however sombre the occasion; and the girl with the perpetual cliché about the nice flowers, or the baby talker with her 'How's your little tumsie wumsie today, Dad?' The Royal College of Nursing might employ a Miss Ruth Draper in this form of tutoring. But we must not be too severe, for we know from bitter experience that occasionally patients can be very ugly customers.

Other exercises are in the use of telephones and the interview of relatives. In hospitals we know how tiresome the inquirer may be, but they, the tiresome inquirers, are probably fewer than 5 per cent., and we must adjust ourselves to the other 95 per cent. of earnest, harassed people who come to hospital for news. To have earned from them the heartfelt thanks for a courtesy and sympathy they have never seen equalled before is a nurse's finest hour. And,

in spite of all the criticism of hospitals, that does happen. Indeed, when I look back and around I see that some of the wisest, most courteous, and most humane of human beings I have ever known have been nurses with whom I have worked. They have had a basic understanding, an expert understanding, and a specialized understanding of the individuals entrusted to their care. But for the sake of the young nurse I think we might do a little more about it.

Perhaps I have made it sound too easy, when we know that in spite of example and training many of us blunder through life without ever learning to understand the individual. I am not thinking of the near-moron type in which the defect is obvious. There are others with intelligence and energy who fail to understand. Every time they open their mouth, they put their foot in it, so to speak. Fortunately they do not get very far in the practice of medicine, and do not often reach posts of responsibility in the *practice* of nursing. At least they do not if the patients have a free choice of doctor. In these circumstances doctors lose their patients. We recognize this type of person as a rather blatant but incorrigible ass.

There is, however, another type who also does not fit well into the practice of medicine, but is more difficult to recognize. He is intelligent enough, but is insensitive to other people's feelings. He understands individuals only too well, and if I call him the 'confidence trick man', or the charlatan, I do not need to describe him.

The danger is that these two types, failing in the practice of medicine or nursing, may drift into administrative or other employment where, although an understanding of individuals and their feelings is just as necessary, their defects are not so readily exposed. They become crooked pegs in straight holes. If you agree that these types are not fitted for medicine or nursing, perhaps some of you will tell me how to keep them out, or straighten them out. But here again we return to our original difficulty, which is not of understanding but of knowing what to do.

In one field the work of doctors and nurses comes near to that of teachers. That is in the understanding of a child. It may be of material benefit if we here compare our methods and opportunities. On my part, when I need to understand a child or his illness in order to advise what shall be done for his welfare and treatment, I would hesitate to reach an opinion without a close scrutiny of the

parents, without listening to their opinions, and without a knowledge of the conditions in which they live. In some cases it may be necessary to go further than this, and find out, for example, what help a harassed young mother may have at hand in the way of neighbours and grandmothers. *This is not sentimental twaddle.* It is a biologist's approach to the understanding of a young individual. It is fashionable now to call it the ecological approach.

In the practice of medicine we seek this aid by consultations with the parents as often as possible. Such understanding as I have about the young has been greatly enriched by this close contact with parents from whom I have learned so much. 'The heart has its reasons, which the reason knows nothing of', said Pascal. It is important when considering the welfare of children that these reasons should be understood.

With sincerity I think that in this technique the medical profession has something to teach the teachers. If the child is to be committed to a hospital, especially to a long-stay hospital, we would not readily make a decision about the type of hospital to which the child should be sent without this interview with the parents, and without this assessment of their responsibility. Therefore, it strikes me as somewhat arbitrary the way in which a child's educational future and pedagogic treatment is prescribed without such interviews and sometimes without an interview of the child himself. His educational future then depends on an impersonal examination paper or intelligence test, which strikes me as not a very individual line of approach. But here I am transgressing into territory which is not my own, not knowing whether these tests and papers are sufficient to reveal a basic understanding, an expert understanding, and a specialized understanding of the child.

XIX. *The Methodology of Clinical Science*¹

THERE is a risk in generalizing from the few facts which we apprehend in our personal experience, and I take this risk in saying that there are two main kinds of clinical science, of which one is in danger of being overwhelmed by the other. Out of this distinction I shall try to create the substance of this lecture.

Clinical science is the planned study of disease, or of the phenomena of disease, in men and women and children who are sick. It is a practical craft, and this implies that the clinical scientist must possess the particular skills necessary for collecting and systematizing his knowledge. He must possess also the opportunity to do his work with such regularity and method as the occasion demands. Working within the responsibility for the medical care of the patient, he must do nothing which is harmful to his patient or which delays his cure. If his researches lead the clinical scientist to do more than this, or less than this, his intentions must be approved by the patient after full explanation of what is in store for him. Approval by the patient will always be influenced by the attitude and the opinion of the investigator, and there can be no tribunal to pass judgement on what is practice and what is malpractice in the investigation of sick patients. Under these circumstances research methods can be sanctioned only by that faculty which distinguishes right from wrong, and which we may still call conscience. This understanding of what is right and what is wrong in the care of patients is part of the professional knowledge gained in medical schools and teaching hospitals from teachers, who hold their teaching posts for the very reason that they can transmit this knowledge, with other knowledge, to their assistants and students.

I return to the two kinds of clinical science. One way of approach to a definition is to look at their aims. The aim of one kind of clinical science is to know disease in such a way as to enable us to infer its cause and to predict its course. Seen in this way a disease is regarded as a sequence of phenomena in a sick person arising from and following on a noxious injury; and this is in accord with the

¹ A lecture to the British Postgraduate Medical Federation, University of London, delivered at the Postgraduate Medical School of London on 6 January 1953. Published in the *Lancet*, 265, 1953, p. 629.

Aristotelian idea of the nature of things. The methodology of this kind of clinical science is the planned observation of the sequence of phenomena in that number of sick people which allows us to know the common course of the disease under study from its beginning to its end, and to estimate and know the variations from that course. Planned observation implies the use of all practicable and necessary machines of measurement. The knowledge it leads to has immense practical values, for to know disease in this way is the basis of exact therapy in professional practice. This kind of work might be called clinical cartography, because it helps the practitioner to know where he is, and where he should go.

The other kind of clinical science might be called clinical phenomenology. It studies the phenomena of disease or of disordered function, and its aim is to explain their mechanisms and their clinical significance. It is concerned with auricular fibrillation, hunger pain, itchy skin, or hyperpotassaemia, rather than with the diseases in which these phenomena arise. Its methodology starts with identification of the phenomena and then goes on to the use of experiment when possible and necessary. Great benefits have come to both the science and practice of medicine by these detailed analytical studies of clinical phenomena. The knowledge gained has both increased our understanding of old diseases and pointed the way to the discovery of new diseases.

Thomas Lewis did so much to establish this precise and rational study of clinical phenomena that it is tempting to use his name as an eponymous title for this kind of clinical research, but Lewis's view of medicine went far beyond detailed studies of phenomena, and for that reason I have suggested that, if a name has to be found, it would be better to call it clinical phenomenology. Thomas Lewis, however, by the mere force of his personality has so influenced clinical research in this country in recent years that the study of clinical phenomena, or 'states of disease' as he called them, overshadows other kinds of clinical research. The advantages derived from this influence have been enormous, but there are grounds for believing that its dominance has led to the neglect of the planned study of disease.

CLINICAL OBSERVATION

Before I go further I must try to make clear the different kinds of clinical observation. That the instruments of clinical observation

are now so disconcertingly numerous makes this all the more necessary. We should keep in mind, however, that it is the essence of clinical research that, while the observer should know what to look for, he should not be too absorbed to notice the unusual. To grasp the significance of the unusual is an uncommon quality, and F. M. R. Walshe stressed this idea in saying:

It takes the exceptional mind to pass beyond the first exercise of the discriminatory tendency to pick up a hitherto unrecorded phenomenon and to grasp its significance. Indeed, many a competent clinician may live out his professional existence, never having made a truly original observation.

With this chastening thought in our minds we can try to separate clinical observation into its parts.

There are three main types of clinical observation, and each is as much an expression of a natural cast of mind as of a trained method of approach; which is not to say that they cannot all be combined occasionally in one person's skill. How occasionally I cannot estimate, for I cannot recollect having seen such a person.

The first type of observation is the episodic recognition of spontaneously occurring signs of disease which we use when we diagnose and prognosticate. It is the craftsman's skill in seeing quickly what he knows to be significant, and, as such, it grows out of experience. Not all are capable of it to an excellent degree. It comes best to those who talk least, and the ebullient loquacious teacher rarely possesses it. It sees the rose spot and the scabies burrow. It hears the cry indicating a retropharyngeal abscess, and the wheeze of tracheal pressure. It knows when to dismiss the record of a high blood-urea as a laboratory mistake or as an error in collecting the blood into a contaminated tube. In its best form it works towards an economy of effort, and has its eye on what is true and what is false in new machines. To this practical skill of the experienced clinical observer it is customary to give the name of clinical intuition; but if this implies a kind of mystical guessing, it no more deserves the name than does the skill of knowing where to find a plover's nest. It is the product not of guessing but of a sifted experience by which the significant is recognized with such rapidity that the steps of reasoning are not discernible to the uninitiated. There is, of course, and all too commonly, such a thing as clinical guessing, which we may suspect when we hear it

bolstered up by expressions of opinion beginning with the words 'In my experience . . .'; but this does not come into any of the categories of serious clinical observations we are discussing.

This episodic or incidental type of clinical observation is not in itself a method of research. It is the tool of professional practice. However skilfully it may be used, it is not likely to lead either to discovery or to a decisive clarification of existing knowledge, both of which are the objects of research. The truth of this will be known to those who have used the routine clinical notes of hospitals in seeking recorded facts and observations. These records may have their face value as a teaching instrument, but as instruments of clinical research the routine ward notes and the machinery of hospital records are almost useless.

The second type of clinical observation is so rare that I may not make it evident except in examples. It comes only to the mind prepared. It is Coleridge's 'ascertaining vision' which comes to a few 'who even in the level streams have detected elements which neither the vale itself or the surrounding mountains contained or could supply'. Its essence is the recognition of a new phenomenon or of a new correlation between two previously recognized phenomena. Coming to those with a sufficient technical experience, it takes shape only in the minds of those endowed with imagination and insight. It is near to the artist's power to see something fresh in 'things we have passed a thousand times nor cared to see', but it has been an attribute also of most of the great scientists. To describe it I will mention only the example of N. M. Gregg's brilliant recognition of the association between rubella in the pregnant woman at her eighth to tenth week of pregnancy and the congenital deformities in the child who will be born 7 months later. If Gregg had not systematized his observations quickly at that time, and in those circumstances, the chance might not have come again, or for another hundred years or more. It was fortunate that the chance came to the sensitive mind. This kind of clinical observation, for which I can find no suitable name, is one of the most scintillating starting-points of scientific research. But we cannot order it or command it. The best we can do is to recognize its possibility in the sensitive few, and to prevent its inhibition by too much teaching, its submersion by too much dogma, and its extinction by too much ritual even though it be the ritual of research.

The third kind of clinical observation is what we seek to cultivate

in clinical research. It is clinical observation planned to answer a carefully prepared question. The plan is controlled by well-known criteria: (a) that the question is worth answering; (b) that it has not already been answered by someone else; (c) that a plan of observation can be designed which is likely to provide an answer to the question; and (d) that the observer is the sort of man who will carry out the plan. This kind of planned observation is one of the established methods of clinical research, and its scientific value is judged by the design of the plan. Its purpose is to discover something, or to clarify some already existing field of knowledge. In clinical medicine the latter is not less important than the former; for growth of knowledge is not inevitable, and there is, I think, such a thing as a constant growth of ignorance in medicine, which can be kept in check only by opposing to it the clarifying process of research into the validity of existing knowledge.

Examples of planned clinical observation appear in various forms, but I choose the investigation of the outbreak in 1948-9 of poliomyelitis in Canadian Eskimoes at the Chesterfield Inlet because it shows how observation may on occasion rise superior to the experimental method. If Peart, Rhodes, and their colleagues had not taken the opportunity to plan and carry out this observation the circumstances might not have occurred again; nor could anyone have designed an experiment to repeat the circumstances. Its significance lay in the fact that it was an outbreak of poliomyelitis in a virgin-soil community, the like of which, with the advent of the aeroplane, we are not likely to see again. The picture was unique. It showed a case-incidence of paralysis of 185 per 1,000 of the population; 1 in 20 of the population died; no children under the age of 4 years were paralysed; and the epidemic reached its height in February with the mean temperature at -31°F . Here was something unparalleled in the recorded epidemiology of the disease, and the success of the observations depended on the team of five medical men who flew from Toronto to the scene of the epidemic and carried their plans into effect. There are many other examples of planned clinical observation, in more obvious fields of inquiry such as controlled therapeutic trials and studies in prognosis; but the essential features of all are the choice of question worth answering, the definition and limitation of what is to be observed, the training and character of those who will make the observations, and the arrangements by which the material for

bolstered up by expressions of opinion beginning with the words 'In my experience . . .'; but this does not come into any of the categories of serious clinical observations we are discussing.

This episodic or incidental type of clinical observation is not in itself a method of research. It is the tool of professional practice. However skilfully it may be used, it is not likely to lead either to discovery or to a decisive clarification of existing knowledge, both of which are the objects of research. The truth of this will be known to those who have used the routine clinical notes of hospitals in seeking recorded facts and observations. These records may have their face value as a teaching instrument, but as instruments of clinical research the routine ward notes and the machinery of hospital records are almost useless.

The second type of clinical observation is so rare that I may not make it evident except in examples. It comes only to the mind prepared. It is Coleridge's 'ascertaining vision' which comes to a few 'who even in the level streams have detected elements which neither the vale itself or the surrounding mountains contained or could supply'. Its essence is the recognition of a new phenomenon or of a new correlation between two previously recognized phenomena. Coming to those with a sufficient technical experience, it takes shape only in the minds of those endowed with imagination and insight. It is near to the artist's power to see something fresh in 'things we have passed a thousand times nor cared to see', but it has been an attribute also of most of the great scientists. To describe it I will mention only the example of N. M. Gregg's brilliant recognition of the association between rubella in the pregnant woman at her eighth to tenth week of pregnancy and the congenital deformities in the child who will be born 7 months later. If Gregg had not systematized his observations quickly at that time, and in those circumstances, the chance might not have come again, or for another hundred years or more. It was fortunate that the chance came to the sensitive mind. This kind of clinical observation, for which I can find no suitable name, is one of the most scintillating starting-points of scientific research. But we cannot order it or command it. The best we can do is to recognize its possibility in the sensitive few, and to prevent its inhibition by too much teaching, its submersion by too much dogma, and its extinction by too much ritual even though it be the ritual of research.

The third kind of clinical observation is what we seek to cultivate

phenomena subjectively interpreted by the patient and objectively identified by himself. His main task is to place the phenomena in temporal and in quantitative relationships with each other. This leads him to know the course of a disease as it may be expected commonly to occur. His next task is to determine the variations from that course and to find correlations between these variations and aetiological factors or alternative treatments. When possible he uses statistics to express these variations. He uses statistical estimates of variations also in designing the extent of his study. If the disease under study is one which varies little in its course, he limits his number of examples. If the course of the disease has many variations—or a wide ‘lunatic fringe’ as the clinical scientist may call it in his despondent mood—he takes this into consideration in making his estimate of the number of patients to be taken into his study, and then proceeds towards that goal. If he takes less than he needs, his study is deceptively incomplete. If he takes more than he needs, he wastes his time. In this way he plans his study of disease, and with one research finished he passes on to study another disease. In this way he comes to know disease as a predictable sequence of events, and the knowledge gained becomes the basis, the only basis, by which the underlying processes of disease in the living patient can be rationally interpreted.

I may be setting my aim too high in describing this method of study, or this method of study may have less scientific value than I think it has; but assuming that it is a necessary and valuable instrument for one kind of clinical research I believe it to be greatly neglected. Apart from a few elementary observations on pulse and temperature, how little we know about the detailed course of disease. How rarely does a clinical investigator sit through an illness recording the observations and instrumental measurements for himself. How seldom does the young physician sit through a night recording the pattern of behaviour, of delirium, or of sleep in a patient with encephalitis. How infrequently does the young surgeon train himself in diagnosis by sitting at the bedside of a man with ruptured duodenal ulcer to measure the fluctuation of his signs and symptoms during the period while the operation is prepared. If the illness be prolonged how rarely do we design our observations with a regularity of 12-hourly or even daily intervals. Most of us come to the patient to observe only the phenomena which custom has predetermined for us, or we take the observa-

tions second-hand from others who have recorded them for us. I am conscious of this in confessing that I myself know very few diseases in this discriminating manner which satisfies my scientific conscience. For example, I could not, to my own satisfaction, describe measles, or leukaemia, or acute rheumatism, or primary herpetiform stomatitis, with their hour-to-hour or day-to-day sequence of significant phenomena precisely determined, measured, and accurately recorded. Yet this kind of knowledge is one of the bases on which scientific clinical medicine is built. It may not reveal and unfold any new natural laws, but the practice of rational medicine relies on it; for, without the power to predict the natural course of a disease, advice and treatment will remain at the mercy of insubstantial opinions.

My claim is that clinical science should correct the bias which has been imposed upon it by dominance of the experimental study of phenomena, and that a few young clinical scientists should be trained in the exact observation and measurement of disease. If it be asked what value can these have, since diagnosis and treatment are the end-all and be-all of practical medicine, my reply would be that it would bring mature and trained observers to the bedsides of patients and that in those circumstances new significant phenomena are likely to be discovered. Even if no new discoveries be made in this way, it will at least result in a contemporary knowledge of disease and illness, for the pattern of these, changing as they do in the new environments which man creates for himself, are not static and eternal.

The inaccuracy and inadequacy of our recorded knowledge about disease and illness will become evident if we consult a few examples in textbooks and journals. Nowhere in medical literature can I find a complete coherent account of the sequence of clinical events which follows the burnings of a man, a woman, or a child. If convulsions occur after burning, is their frequency related to the age of the patient or to the extent of the burn? Is the time-incidence of the convulsion, if it does occur, limited to the second day of the illness? There is no recorded answer to these questions. Many studies of isolated phenomena of burns have been successfully pursued, but the close relationship of one phenomenon with another has not been established. That this should be the case in a disease so easy to observe and to measure is an index both of our ignorance and of faulty observation.

On the other hand when we hear a young clinician say, after examining and observing a suspected case of meningococcal septicaemia, 'I have been watching the appearance and measuring the changing outline of these purpuric spots over the last six hours, and from this evidence I know that this illness is not meningococcal septicaemia because this illness is at its fiftieth hour, and the purpuric spots of meningococcal septicaemia are not that shape at this hour of the illness'—when we hear him describing his observations in that way we know that he is heading in the direction of being a clinical scientist. But there are many hindrances to this training and many obstacles to that kind of clinical work, which I must now attempt to discuss.

HINDRANCES

This talk about scientists may build them up too much. No sensible young man starts out to be a clinical scientist. By accident or opportunity he later finds himself heading in that direction and follows the stream; or he comes under the personal influence of someone he likes and wishes to imitate; or he is just that sort of chap who must do research because he has a passion for getting things clear in his own mind, or for seeing how things tick over, and finding no other outlet or way of doing it he tries his hand at clinical research. He equips himself to become a clinical scientist and then he is between the physical scientists, who do not understand what he does, and the practitioners of medicine and surgery, who try to understand what he does. He seeks a job either as a full-time professional research-worker, or by combining research with some routine clinical practice. The latter is the more difficult path to follow, particularly in that clamouring kind of hospital which is lively with many conferences and other distractions. But he may order his life successfully by arranging clearly demarcated periods or days, in the way that Victor Horsley did, when he collects and systematizes his observations.

Hindrance to clinical research lies less in the claims of other clinical responsibilities than in the prevailing attitude of mind towards it—the climate of opinion, as it is now called. This attitude of mind begins to take shape in medical schools, where the undergraduate thinks of medical research only as an affair of animal experiment and laboratories. (I must exclude from this generaliza-

tion the three, or perhaps four, *alpha* schools in this country which present a more wholesome picture of research.) In the teaching hospitals he is immediately engaged in the excitements of diagnostic medicine, and his teachers are those whose skill is mainly in that branch of medicine. He runs into the danger of confusing clinical science with something called 'clinical investigation', and of regarding scientific method as synonymous with the use of diagnostic machinery. These misleading impressions are intensified by his preparation for final examinations with their diagnostic exercises as 'long cases' and 'short cases'.

These impressions about the attitude towards clinical research are strengthened by my experience on a regional hospital board committee which reviews applications for research grants from members of the medical staffs of the hospitals. The applications come almost entirely from members of the clinical staffs, and they fall roughly into four categories. The majority of applications are for the employment of morbid anatomists, or biochemists, or technicians to do some laboratory research which the clinicians wish to have carried out but in the techniques of which they themselves have had no training. The second category consists of requests for secretarial help in retrospective studies of hospital records. The third category is the request for diagnostic machinery. The smallest category is the request for the assistance of a clinical research-worker to enlarge and complete a piece of clinical research which is already under way, and which the applicant is conducting for himself.

With moneys now available from the universities, from endowment funds of hospitals, from the government research councils, and from the educational trusts, the opportunities for clinical research are now beyond the dreams of what might have been considered possible thirty years ago. We can therefore look forward with great interest to the directions in which these forces will flow. Human nature being what it is, and human institutions what they are, we must expect that the opportunities will attract those who regard the publication of a research paper as a kind of higher diploma which will increase the chance of their being promoted to a higher clinical post. Others will use the opportunities to get from these resources the extra help they need for their routine clinical work in hospitals. These frailties can be easily humoured and helped in other ways than by calling them research; but if they

must be called research then we must find another name for the kind of work a man does when he devotes himself to the planned and patient observation of disease, which is guided by a substantial working hypothesis.

Behind all these opportunities and arrangements I see the urgent need for a due proportion of young doctors who will become physicians and surgeons and family practitioners to be trained in this kind of work: otherwise we shall face the position of which E. D. Adrian warned us in 1952:

The pathologists and biochemists will find [he said] that their time is taken up with measurements of uncertain value in which they are not specially interested and the final result may well be that the work is turned over to specially trained technical experts who are the last people to give a dispassionate judgement on the value of what they are doing.

He went on to indicate the cure for this in saying: "The point, surely, is that if such data are to be valuable there must be the right people to consider them."

We are now concerned with the selection, the training, and the working conditions of these right people. This brings me back to where I started. We are not likely to lack a sufficient number of the right people trained in the study of isolated clinical phenomena and their mechanisms. The influence of the schools of physiology and experimental pathology will continue to work in that direction. But I greatly fear that the very complexity of these phenomena is withdrawing interest from the planned study of their causative diseases. To that extent there is a growth of ignorance about these diseases. The tempo of events in some diseases may be so slow that they will be accurately observed only by a general practitioner, or by a family doctor over a period of many years; or the disease may be so uncommon, and its distribution so wide, that special arrangements may be required to gather a sufficient number of examples within the reach of the observers. Hospitals as we know them will not be the best places from which this work can be organized. If clinical research is to be used to get a full picture of disease, it must equip itself to carry the observations beyond the hospitals and extend the researches which can best be done in family practice and by field survey of random samples of population. The methodology of planned clinical observation in family practice and in random samples of the population will become a responsibility of medical schools.

I foresee the day when medical faculties of universities will have three provinces of clinical training and clinical research. One will be in hospitals, where the training and research will be dependent on the facilities which remain after the demand for specialist forms of treatment have been satisfied. The second province will be a sample of population in their own homes which can be provided in family practice. The third will be in expeditions to communities, whether they be in a mining village, a Hebridean island, a Chesterfield Inlet, or wherever the inquiry takes them. If we are not too pompous about it all; if we can escape the dead hand of dull conformity; if we can get the freedom to make experiments in arranging our institutions; if we can recover the initiative to make the experiments—then our medical schools and teaching hospitals may take on this work. If they fail it is difficult to see how else it can be done.

XX. *Institutional Medicine*¹

IT is, I think, one of the inherent peculiarities of an Englishman, and more particularly of a Northumbrian, that if you scratch him he begins to brood over his institutions. And then from time to time he reforms them. He is, as it were, still in the atmosphere of those tremendous years before and after 1688, when our revolution was settled more by sincerity of argument than by force of arms, and when there were men of the calibre of Halifax the Trimmer to hold the balance. Now, again, in this century we are in the midst of another revolution, which is shown by our concern about our institutions. If we have neither the vigour, which is an affair of the spirit, nor the means, which is an affair of the intelligence, to reform our institutions, it will be a matter of interest mainly to the historians of the future; but such a state of affairs will be cold comfort to those of us who believe that the excellence of a civilization is shown in the capacity of its citizens constantly to reform its institutions in order to meet its changing cultural needs. But change may be for the better or the worse, and in remembering this we should be guided also by Burke's admonition, that 'merely to innovate is not to reform'.

To be an institution means many things, but on the whole the word has now a derogatory meaning. It conjures up pictures of buildings and committee rooms, of barracks and corridors, of orphanages and museums; and to call someone an 'institutional child' is to speak half in pity and half in reproach. I will try to refresh the meaning of the word by reminding you that an institution is people and not things. An institution is the establishment and arrangement of human effort towards a common end. It will stand or fall according to the value of this end, or purpose as we may call it, and according to the means which are used to reach it. The means are the Government, the administration, and the arrangement for carrying out its work—three quite separate

¹ The opening passages of the presidential address to the Newcastle upon Tyne and Northern Counties Medical Society delivered in October 1953, followed by an outline of the rest of the lecture, prepared from Sir James Spence's notes. Published in the *Lancet*, 263, 1955, p. 41.

functions which must not be confused. As the end never justifies the means, so these means or functions are all-important. By these standards most institutions neither stand nor fall, but just flounder along, because *their end is forgotten, or because their means are confused or inept*. You must forgive my presumption in placing these elementary thoughts in your minds before turning to the profession and practice of medicine which is now so involved in institutions that *I must attempt to find a way through their maze*.

Seeking definitions for the foundation of an argument like this, I go back to that early arch-Englishman Thomas Hobbes—who was 'rare at definitions'. In discussing how the liberty of subjects is to be measured, he stresses the overriding need for government, or sovereignty as he calls it, and distinguishes between Sovereignty by Acquisition, and Sovereignty by Institution. The former is to govern through the seizure of power; the latter is to govern by the *covenant of one to another, or of the many to the few*. This definition gives to the word institution the meaning I wish to convey. Institution is to covenant that something shall be carried out and to transmit to someone the authority to arrange for it to be carried out. I shall accept this definition when I mention the institutions which affect medicine and the disorders which beset them.

I have indicated that institutions may be good or bad, or good in parts, or just floundering along. If they are bad there are usually three possible causes. These are (1) that they do not know or see clearly what they are trying to do—or there are conflicts of purpose in what they are trying to do; (2) that they have not the will to do what they know they should do (which is a fault of government); and (3) that they do not have the ways, and kind of people, to do what they want to do (which is a fault of arrangement or administration). Where arrangement or administration fails it is usually because *there has been no clear definition of responsibility*.

The simpler the institution, and the clearer its purpose, the easier it is for it to be successful. At one end of the scale we have simple institutions, like football teams, cricket clubs, ships' crews, and public schools, whose purposes are clear, and in which the division of responsibility is precise under arrangements which can be judged by results. At the other end of the scale we see complicated institutions like trade unions, professional associations, regional boards, trade corporations, national coal boards, and city councils. If they fail or flounder it is because *their purpose is not*

clear or because their government and arrangement do not match their purpose.

[Here Sir James spoke of the definitive distinction between a service and an institution.]

I can go no further in this attempt to anatomize human institutions, but before I look at the institutions which affect medicine I will recapitulate in mentioning three aspects of them which may be worth bearing in mind.

The first is the great increase in number and size of institutions in recent years. This carries the danger that we are creating institutions beyond the capacity of the available intelligence, the available will, and the available morality which are necessary to maintain them.

The second point, which arises from the first, is that the creation and maintenance of complicated institutions demand a particular kind of intelligence, personality, and education in those responsible for them. The form of this education took shape in John Locke's famous essay, and the substance of it was given in our older universities, at a time when they were designed to give an education for responsibility. This education was reinforced by a system in which the younger men knew intimately the older men experienced in the art of government. In the aristocratic tradition this was still further strengthened by the concept of *noblesse oblige*. If we were to seek modern examples of human institutions we could use John Anderson in our own day, and Robert Morant before him; but the weakness of our present situation is that it is impossible for a sufficient number of people to get close enough to men like Anderson and Morant to learn from their examples.

The third point concerns the manners of those who take part in the affairs of institutions: and by manners I mean *mores*, and therefore morality. It is beyond question that the manner, or morality, of most of us towards a simple institution like our family or our cricket club tends to be of a different order from that we adopt towards complicated and impersonal institutions like British Railways or the Commissioners of Income Tax. An appropriate morality is achieved satisfactorily in a good naval vessel, a good battalion, and a good university; but in the vast impersonal social institutions we are now creating the very opposite may happen. Perhaps our fate is that our civilization will flounder in the midst of the vast institutions it builds, but which it cannot maintain, for

want of capable men. They will then be like the dinosaurs—too big for their jobs, or too big for their shoes.

[There was need, Sir James continued, for careful study of the defects and disorders of the many kinds of institutions affecting the profession and practices of medicine—for the study of *clinical institutology*, or the *morbid anatomy of medical institutions*.

Explaining what he meant by *medicine*, he described it as the fulfilment of a social contract or covenant between a professional man and another man who is sick or who fears he is sick. The fulfilment of this contract was in giving advice, explanation, or treatment, or any combination of these. The contract might be with a group of professional men, but in that case there was clear division of responsibility. Personal responsibility was the essence of medicine—the patient goes to a doctor, not to a board of governors or a clinical cure company—and the doctor's first responsibility was to his patient, not to his employer.

TYPES OF INSTITUTION

Among the institutions affecting medicine two kinds of professional association could be distinguished: (a) those which are simply medical societies, and (b) those which have power over other people, such as the Royal Colleges with their diplomas and the specialist societies in America.

Of these the medical societies came first, for many reasons. Of all medical institutions they were the most moral—in purpose, in method of government, and in method of administration. They had frailties rather than disorders, and these were all in the officers rather than the members.

A third type of medical institution was family practice, whether by a group or by a single doctor. Fourth, we might have the health centre—but not yet. He thought this would become a local service rather than an institution.

As an example of an institution nearly perfect in type, he described the working of the Medical Research Council and its National Institute of Medical Research.

Turning finally to the hospitals, he spoke of their development in four phases: first, the founding phase (about 1750); secondly, the period when the hospital was the place of the predominant

practitioner (1800-1900); thirdly, the period when it was the jumping-off ground for consultants—when practice was obtained through hospital reputation (1900-30); and fourthly, the period when it became a centre for specialist services, with development of peripheral specialist units.

Of hospital problems, the first was size: a hospital of up to 150-200 beds could be an institution, but above that size it was likely to become a service. In speaking of organization, he recalled his *definition of institution as the establishment and arrangement of human effort towards a common end*, and said that he knew of no example of a good hospital which has not been created through the good offices and goodwill of its medical staff. Occasionally one might see a hospital which is very good but in which the medical staff appeared to play very little part in the government; but such a hospital functioned through the excellence of its component institutions. It was a fault if the medical staff either cannot or do not give guidance to the governing body.

The size and complexity of the large modern hospital made it a service rather than an institution, and much depended on the size of its constituent units. The size of the unit in turn depended on the proper size of the team in charge of it, which (he suggested) should not exceed 10-12.

The task of the teaching hospital was complicated by its additional responsibility for teaching and research; and here Sir James expressed his regret at the failure of the concept of the region with a *university hospital at its centre*. *The position of the teaching hospital had become precarious through the growth of regional hospitals as specialist centres and through its consequent loss of acute medicine and surgery and the diseases about which general practitioners most need advice and help*. *In his opinion it had been a grave mistake not to make the teaching hospital a university institution, giving it a local area in which it would be responsible for designing the entire hospital service*. It should be a central hospital for acute medicine, surgery, and fevers, and with specialist annexes.

THE UNIVERSITY PATTERN

Outlining the design of the teaching hospital—its methodology, its morality, its style—Sir James drew a parallel with the English

university at its best. Its three purposes—clinical science, clinical teaching, and the care of the sick—were comparable to the three purposes of a university, which are to advance scholarship, to teach students, and to create a university life; and there need be no conflict between these purposes. He believed that, just as universities are free from the control of the Ministry of Education, so teaching hospitals should be free from the control of the Ministry of Health.

In conclusion, he spoke of his faith in the future of our universities, through which England would endure. 'They are big but they remain an institution.' Conversely, he had great fears for the future of hospitals, and foresaw a day when the best of our young men will go into simpler and more satisfying institutions—notably general practice.]

BIBLIOGRAPHY OF THE WRITINGS OF JAMES SPENCE

1920

1. 'Some observations on sugar tolerance', *Quart. J. Med.* 14, 1920-1, pp. 314-26.
2. 'Acute rheumatism in children under 12 years' (with F. J. Poynton and D. Paterson), *Lancet*, 199, 1920, pp. 1086-9.
3. 'Oxycephaly' (with R. C. Jewesbury), *Proc. R. Soc. Med.* 14, 1920-1. Sect. stud. dis. childh., pp. 27-30.
4. 'Acrocephaly with other congenital deformities' (with R. C. Jewesbury), *Ibid.*, pp. 30-35.

1921

5. 'The after-effects of epidemic encephalitis in children' (with D. Paterson), *Lancet*, 201, 1921, pp. 491-3.
6. 'The use of Laevulose as a test for hepatic inefficiency' (with P. C. Brett), *Ibid.*, pp. 1362-6.
7. 'The value of blood sugar estimation in clinical diagnosis.' Thesis accepted for the degree of M.D. (Durham), 1921, unpublished.

1925

8. 'Mental sequelae of encephalitis lethargica', *Lancet*, 208, 1925, p. 280.
9. 'The treatment of spasmophilia', *Ibid.* 209, 1925, p. 1242.

1927

10. 'Cholaemia: a clinical study of nervous symptoms in liver atrophy' (with A. G. Ogilvie), *Arch. Dis. Childh.* 2, 1927, pp. 41-48.
11. 'Significance of occult blood in stools', *Neur. med. J.* 7, 1927, pp. 122-8.
12. 'Diffuse sarcomatosis of brain and spinal meninges' (with R. P. Smith), *Ibid.* 8, 1927, pp. 47-51.

1928

13. 'The liver and pernicious anaemia', *Neur. med. J.* 8, 1928, pp. 71-81.
14. 'Some medical emergencies in childhood', *Practitioner*, 121, 1928, pp. 384-90.
15. 'Chronic nephritis in childhood', *Brit. med. J.* 2, 1928, pp. 1124-8.

1929

16. 'Albuminuria', *Lancet*, 216, 1929, pp. 623-4.

¹ Abbreviations of periodical titles are those given in *World List of Scientific Periodicals*, 1952.

1931

17. 'Clinical study of nutritional xerophthalmia and night blindness', *Arch. Dis. Childh.* 6, 1931, pp. 17-26.

1932

18. 'Child with pink disease who chewed his tongue off', *Newc. med. J.* 12, 1932, pp. 39-42.
 19. 'Benign tuberculous infiltration of the lung (epituberculosis)', *Arch. Dis. Childh.* 7, 1932, pp. 1-8.

1933

20. 'Clinical tests of the antirachitic activity of Calciferol', *Lancet*, 225, 1933, pp. 911-15.
 21. 'Ward and dormitory infections', *Ibid.*, pp. 1306-9.
 22. 'Neo-natal diseases', in PARSONS, Leonard G., and DARLING, Seymour G., eds., *Diseases of Infancy and Childhood*, London, Oxford University Press, 1933, 1, pp. 103-36.
 23. 'Disorders of metabolism', *Ibid.*, pp. 631-47.

1934

24. 'Ward and dormitory infections, excluding common exanthemata' (with J. A. Glover and E. K. le Fleming), *Trans. med. Soc. Lond.* 57, 1934, pp. 75-102.
 25. *Investigation into the Health and Nutrition of Certain of the Children of Newcastle upon Tyne between the Ages of One and Five Years*. Newcastle upon Tyne, City and County of Newcastle upon Tyne, 1934.
 26. 'Diseases of the genito-urinary system', in GARROD, Sir Archibald Edward, ed., *Diseases of Children*, 3rd ed., London, Edward Arnold, 1934, pp. 417-52.

1936

27. 'Rickets', *Practitioner*, 136, 1936, pp. 196-202.

1937

28. 'Discussion on difficulties of nutritional assessment' (with H. E. Magee and G. C. M. M'Gonigle), *J.R. sanit. Inst.* 58, 1937, pp. 61-75.

1938

29. 'The modern decline of breast-feeding', *Brit. med. J.* 2, 1938, pp. 729-33.

1939

30. 'Modern views on tuberculosis in childhood', *Practitioner*, 142, 1939, pp. 421-8.
 31. *Report of an Investigation into the Causes of Infantile Mortality in Newcastle upon Tyne during the Year 1939* (with F. J. W. Miller). Newcastle upon Tyne, City and County of Newcastle upon Tyne, 1939.

1940

32. 'The nation's larder in wartime: feeding of children', *Brit. med. J.*, 2, 1940, pp. 93-95.

1941

33. 'The nature of disease in infancy' (Bradshaw lecture abstract), *Lancet*, 240, 1941, pp. 777-80.

1942

34. *Paediatrics in Universities and Teaching Hospitals*. Memorandum for the trustees of the Nuffield Provincial Hospitals Trust. Newcastle upon Tyne, North Eastern Regional Hospitals Advisory Council, 1942.

1946

35. 'Nurseries and welfare of children', *J.R. sanit. Inst.* 66, 1946, pp. 323-6.

1947

36. *The Purpose of the Family*. (A guide to the care of children.) The Convocation lecture 1946, of the National Children's Home. London, Epworth Press, 1947.
37. 'The care of children in hospital' (The Charles West lecture), *Brit. med. J.* 1, 1947, pp. 125-30.

1949

38. 'Medical diseases of the genito-urinary system' (with George Davison), in GARROD, Sir Archibald Edward, and others, eds., *Diseases of Children*, 4th ed., London, Edward Arnold, 1947-9, 2, pp. 417-52.
39. *The Need for Understanding the Individual as Part of the Training and Function of Doctors and Nurses*, London, National Association for Mental Health, 1949.

1950

40. 'Acute intussusception in childhood' (with Donald Court), *Brit. med. J.*, 2, 1950, pp. 920-1.
41. 'Family studies in preventive paediatrics' (Cutter lecture), *New Engl. J. Med.* 243, 1950, pp. 205-10.
42. 'Effects of socialization on paediatrics', Zurich, *Transactions of Sixth International Congress of Paediatrics*, 1950.

1951

43. 'The Doctor, the nurse and the sick child', *Amer. J. Nurs.* 51, 1951, pp. 14-15.
44. 'Poliomyelitis', in PARSONS, Sir Leonard Gregory, ed., *Modern Trends in Paediatrics*, London, Butterworth, 1951.
45. *The Young Mother at Work*. Report of a conference held in Newcastle upon Tyne, December 1951. Newcastle upon Tyne, Tyne Council of Social Service, 1951.

1931

17. 'Clinical study of nutritional xerophthalmia and night blindness', *Arch. Dis. Childh.* 6, 1931, pp. 17-26.

1932

18. 'Child with pink disease who chewed his tongue off', *Newc. med. J.* 12, 1932, pp. 39-42.
 19. 'Benign tuberculous infiltration of the lung (epituberculosis)', *Arch. Dis. Childh.* 7, 1932, pp. 1-8.

1933

20. 'Clinical tests of the antirachitic activity of Calciferol', *Lancet*, 225, 1933, pp. 911-15.
 21. 'Ward and dormitory infections', *Ibid.*, pp. 1306-9.
 22. 'Neo-natal diseases', in PARSONS, Leonard G., and BARLING, Seymour G., eds., *Diseases of Infancy and Childhood*, London, Oxford University Press, 1933, 1, pp. 103-36.
 23. 'Disorders of metabolism', *Ibid.*, pp. 631-47.

1934

24. 'Ward and dormitory infections, excluding common exanthemata' (with J. A. Glover and E. K. le Fleming), *Trans. med. Soc. Lond.* 57, 1934, pp. 75-102.
 25. *Investigation into the Health and Nutrition of Certain of the Children of Newcastle upon Tyne between the Ages of One and Five Years*, Newcastle upon Tyne, City and County of Newcastle upon Tyne, 1934.
 26. 'Diseases of the genito-urinary system', in GARROD, Sir Archibald Edward, ed., *Diseases of Children*, 3rd ed., London, Edward Arnold, 1934, pp. 417-52.

1936

27. 'Rickets', *Practitioner*, 136, 1936, pp. 196-202.

1937

28. 'Discussion on difficulties of nutritional assessment' (with H. E. Magee and G. C. M. M'Gonigle), *J.R. sanit. Inst.* 58, 1937, pp. 61-75.

1938

29. 'The modern decline of breast-feeding', *Brit. med. J.* 2, 1938, pp. 729-33.

1939

30. 'Modern views on tuberculosis in childhood', *Practitioner*, 142, 1939, pp. 421-8.
 31. *Report of an Investigation into the Causes of Infantile Mortality in Newcastle upon Tyne during the Year 1939* (with F. J. W. Miller). Newcastle upon Tyne, City and County of Newcastle upon Tyne, 1939.

1940

32. 'The nation's larder in wartime: feeding of children', *Brit. med. J.*, 2, 1940, pp. 93-95.

1941

33. 'The nature of disease in infancy' (Bradshaw lecture abstract), *Lancet*, 240, 1941, pp. 777-80.

1942

34. *Paediatrics in Universities and Teaching Hospitals*. Memorandum for the trustees of the Nuffield Provincial Hospitals Trust. Newcastle upon Tyne, North Eastern Regional Hospitals Advisory Council, 1942.

1946

35. 'Nurseries and welfare of children', *J.R. sanit. Inst.* 66, 1946, pp. 323-6.

1947

36. *The Purpose of the Family*. (A guide to the care of children.) The Convocation lecture 1946, of the National Children's Home. London, Epworth Press, 1947.
37. 'The care of children in hospital' (The Charles West lecture), *Brit. med. J.* 1, 1947, pp. 125-30.

1949

38. 'Medical diseases of the genito-urinary system' (with George Davison), in GARROD, Sir Archibald Edward, and others, eds., *Diseases of Children*, 4th ed., London, Edward Arnold, 1947-9, 2, pp. 417-52.
39. *The Need for Understanding the Individual as Part of the Training and Function of Doctors and Nurses*, London, National Association for Mental Health, 1949.

1950

40. 'Acute intussusception in childhood' (with Donald Court), *Brit. med. J.*, 2, 1950, pp. 920-1.
41. 'Family studies in preventive paediatrics' (Cutter lecture), *New Engl. J. Med.* 243, 1950, pp. 205-10.
42. 'Effects of socialization on paediatrics', Zurich, *Transactions of Sixth International Congress of Paediatrics*, 1950.

1951

43. 'The Doctor, the nurse and the sick child', *Amer. J. Nurs.* 51, 1951, pp. 14-15.
44. 'Poliomyelitis', in PARSONS, Sir Leonard Gregory, ed., *Modern Trends in Paediatrics*, London, Butterworth, 1951.
45. *The Young Mother at Work*. Report of a conference held in Newcastle upon Tyne, December 1951. Newcastle upon Tyne, Tyne Council of Social Service, 1951.

1952

46. 'Pink disease', in HORDER, Thomas Jeeves, 1st baron, ed., *British Encyclopaedia of Medical Practice*, 2nd ed., London, Butterworth, 1950-2, 9, pp. 609-14.

1953

47. 'The nursing of premature infants in hospitals', *Enfant*, no. 5, 1953, pp. 395-402.
48. 'The methodology of clinical science', *Lancet*, 265, 1953, pp. 629-32.

1954

49. 'Hospital beds for children: an estimate of needs' (with M. D. Taylor), *Lancet*, 266, 1954, pp. 719-21.
50. *A Thousand Families in Newcastle upon Tyne: An Approach to the Study of Health and Illness in Children* (with W. S. Walton, F. J. W. Miller, and S. D. M. Court), London, Oxford University Press, 1954.
51. 'Neo-natal diseases' (with F. J. W. Miller), in PARSONS, Leonard G., and BARLING, Seymour G., eds., *Diseases of Infancy and Childhood*, 2nd ed., London, Oxford University Press, 1954, 1, pp. 128-203.
52. 'Disorders of metabolism', *Ibid.*, pp. 420-34 and 437-8.

1955

53. 'Institutional medicine: from the posthumous papers of Sir James Spence', *Lancet*, 268, 1955, pp. 41-43.

Index

- Abrams, I., 96.
 Abramson, H., 66.
 Acetone, 118.
 Achlorhydria, 41, 46.
 Acro-dynia, *see* Pink disease.
 Acromegaly, 119.
 Adenitis, 155.
 Adrian, E. D., 291.
 Adynamic ileus of the newborn, 63-64.
 Alcock, O., 143.
 Alkaptonuria, 61.
 Amble, 1.
 Anaemia, 150-1, 152, 154; *see also*
 Pernicious anaemia.
 Anaemia haemolytica neonatorum, 62.
 Anderson, J., 295.
 Anorexia, 78.
 Appendicitis, 239, 257.
 Armand-Dehille, P. F., 58.
 Armstrong, C., 84.
 Armstrong, C. N., 143.
 Arsenic, 42.
 Arterio-sclerosis, 165.
 Arthritis, 68, 69, 120.
 Atelectasis, 49.
 Athenaeum, 19, 23.
 Aycock, W. L., 82, 88, 92, 108.

 Babies' Hospital, 5-6, 7, 12, 16, 72,
 165, 181, 254.
 Bailey, C. V., 113, 115, 117, 118, 119,
 128.
 BAL, 78.
 Bang, I. C., 114, 118.
 Barling, S. G., 300, 302.
 Barlow, Sir T., 206.
 Baskin, J. L., 88, 108.
 Baudouin, A., 114, 128.
 Beattie, T., 4, 40.
 Bedson, S., 3, 14.
 Behaviour of children, 195-9.
 Benedict, F. G., 118, 119, 120.
 Benwell Maternity Clinic, 143.
 Bergmark, G., 121, 128.
 Beri-beri, 50, 152.
 Best, W. H., 66.
 Beveridge, W., 194.
 Bevin, E., 272.
 Biermann, A. H., 88, 108.
 Bilderback, J., 71, 78.
 Bing, H. J., 114, 129.
 Birmingham University, 269.
 Bitot's spots, 49.
 Blackader Lecture, 18.
 Blagdon, 12.
 Blood-sugar, 113-29.
 Bodian, D., 82, 89, 90, 108, 109.
 Boils, 50.
 Bornholm disease, 96.
 Boston Children's Hospital, 17.
 Bourdillon, R. B., 131.
 Bramwell, B., 31, 38.
 Bratton, A. B., 101.
 Breast-feeding, 63-64, 161-73, 189.
 Brett, P. C., 299.
 Brewis, C., 164.
 Bright, R., 28, 36, 38.
 Bristol University, 269.
 British Association, 269.
 British Council, 4.
 British Council, 18.
 British Drug Houses Ltd., 130.
 British Medical Association, 17.
 British Paediatric Association, 10, 15,
 18.
 British Postgraduate School of Medi-
 cine, 268, 281.
 Bronchiectasis, 51, 57, 76, 212.
 Bronchitis, 152.
 Burnet, F. M., 82, 83, 89, 91, 108.
 Burns and scalds, 240, 257, 288.
 Byfield, A. H., 71, 78.
 Calciferol, 7, 129-41.
 Callow, V., 74.
 Cameron, H. C., 66, 70.
 Canadian Medical Association, 18.
 Cancer, 111, 119-20, 127-8.
 Carling, E. R., 15.
 Casey, A. E., 96, 108.
 Cass, J., 66, 69, 70.
 Caughey, J. E., 96, 108.
 Central Health Services Council, 17.
 Cerebral haemorrhage, 59, 61, 67.
 Cerebrospinal fluid, 67.
 Chapin, H. D., 206, 209.
 Charles, J. A., 142.
 Cheek, D. B., 74, 78.
 Chesterfield Infant, 285, 292.
 Child welfare clinic, 6, 143, 209,
 227-8, 264-5.
 Child welfare services, 187.
 Children's hospital, 16, 223, 234-49,
 268-9.
 Hospital beds needed, 252-60.
 Medical staff, 244-5.
 Children's Medical Care Council, 228.
 Cholaemia, 27-31.
 Cholaemic type, 31.

- Cholera infantum, 68.
 Cholecystitis, 67.
 Circumcision, 59, 68.
 Cirrhosis of the liver, 27.
 Clinical observation, 281-92.
 Coeliac disease, 65, 131, 239, 248.
 Cohn, E. J., 39, 40, 44, 46, 47, 48.
 Cole, S. W., 122, 129.
 Coma, 29, 31-33.
 Congenital abnormalities, 60-61.
 Conjunctivæ, 49.
 Conjunctivitis, 75.
 Consultation, 274-7.
 Coryza, 75.
 Court, S. D. M., 16, 22, 215, 217, 301, 302.
 Cowell, W., 12.
 Cowie, D. M., 118, 129.
 Coxsackie viruses, 84.
 Craig, W. S., 66, 70.
 Creatinin, 118.
 Creighton, C., 206.
 Cresyl blue, 40.
 Cretinism, 119.
 Culotta, C. S., 83.
 Cummings, E., 5.
 Curtis, M., 16.
 Curtis Committee, 16, 235.
 Cutter, J. C., 17, 204.
 Cystinuria, 61.
 Czerny, A., 206.
 Dale, H., 14.
 Dalldorf, G., 84, 108.
 Danon, L., 63, 70.
 Darwin, C., 177.
 Davidson, S. W., 55, 132.
 Davison, G., 12, 301.
 Deoxycorticosterone acetate, 74.
 De Wesselow, O. L. V., 115, 116, 118, 121, 129.
 Diabetes, 5, 114-19, 125.
 Diabetic acid, 118.
 Diarrhoea and vomiting, 4, 46, 63-64, 67, 95, 153.
 Dingle, J. H., 219.
 Diphtheria, 66, 239, 254, 257.
 Drummond, H., 40.
 Duncan, M., 143.
 Duodenal ulcer, 28, 30, 32, 287.
 Durham University, *see* University of Durham.
 Dykes, R. M., 219.
 Dysentery, 66, 69, 239, 247.
 Dyspepsia, 55.
 Eck's fistula, 31.
 Edinburgh University, 269.
 Eliasberg, H., 51.
 Elizabeth (*consort of George VI*), 11.
 Elliott, T. R., 35, 38.
 Ellis, H., 166.
 Elmfield College, 1.
 Empyema, 52, 68, 69.
 Encephalitis, 28, 33, 125, 128.
 Environmental medicine, *see* Field inquiries.
 Epinephrin, 118.
 Epituberculosis, 7, 51-58.
 Ergosterol, 50.
 Erythroedema, *see* Pink disease.
 Eumydrin, 64.
 Extensor plantar responses, 35.
 Faber, H. K., 89, 108.
 Family doctors, 207-8, 221, 224-6, 264-5.
 Family life, 174-203.
 Farr, W., 206.
 Fawcett, R., 215.
 Feer, E., 71, 74, 78, 206.
 Feer's disease, *see* Pink disease.
 Feggetter, G. Y., 48.
 Fever hospitals, *see* Infectious disease hospitals.
 Fibrosis, 51, 57.
 Field inquiries, 206-16.
 Findlay, G. M., 4.
 Finkelstein, H., 206.
 Finlay, T. Y., 163.
 Fishbein, W. I., 96.
 Fisher, G., 117, 129.
 Fleming, A., 14.
 Flexner, S., 82, 83, 108.
 Foster, G. L., 120, 129.
 Foster-parents, 195.
 Fox, T. F., 20.
 Francis, T., 87, 109.
 Frank, A., 121, 129.
 Frank, E., 114, 129.
 Frant, S., 66.
 Fredenwald, J., 119, 127, 129.
 Frölich's hypopituitarism, 119.
 Galactose, 115.
 Gard, S., 87, 109.
 Garrod, A. E., 300, 301.
 Gastro-enteritis, 66, 68, 239, 247, 257.
 Geffen, D. H., 88, 109.
 George VI, 11.
 Gillespie, J. B., 63, 70.
 Glasgow University, 269.
 Glossitis, 43, 46.
 Glover, J. A., 300.
 Glucose, 114-16, 119, 121-6.
 Glycerin, 83.
 Glycosuria, 31, 113, 115, 119, 121, 125.
 Görtter, E., 57, 58.
 Goitre, 119.
 Goodenough Committee, 15.
 Goto, K., 115.

- Graham, G., 115, 129.
 Graunt, J., 205, 206.
 Gravey, F. K., 99, 109.
 Great Ormond Street Hospital for Sick Children, 4, 233.
 Greenwood, M., 206.
 Gregg, N. M., 284.
 Griffith, J. P. C., 119, 129.
 Grove, G. H., 119, 127, 129.
 Grulce, C., 165, 171.
 Guthrie, K. J., 67, 70.
 Hamman, L., 115, 116, 129.
 Harry, N. M., 105, 109.
 Harvard School of Public Health, 17, 204.
 Health visitors, 211, 225-7.
 Heart disease, 239, 248.
 Heine, J., 81-82, 84.
 Hemiplegia, 102.
 Henderson, H. E., 119, 129.
 Herniae, 59, 239.
 Hicks, C. S., 74, 78.
 Hill, B., 88.
 Himsworth, H., 14, 19, 20.
 Hirschmann, I. T., 115, 129.
 Hirschsprung's disease, 63.
 Holt, L. E., 206.
 Hopkins, A. H., 115, 118, 129.
 Horder, T. J., 302.
 Horsley, V., 289.
 Horstmann, D. M., 82, 89, 90, 109.
 Hospitals, 233-60, 296-7; *see also* Children's Hospitals; Infectious disease hospitals; Maternity hospitals; Orthopaedic hospitals; Teaching hospitals.
 Housing, 156-7.
 Howe, H. A., 82, 89, 90, 108, 109.
 Howland, J., 206.
 Hubbard, D. M., 74, 79.
 Hume, W., 9, 40.
 Hunter, T. C., 54.
 Hutchens, H. J., 8.
 Hydrochloric acid, 46.
 Hyperglycaemia, 37, 113-18, 123, 127-8.
 Hyperplasia, 30.
 Hypotonia, 71, 75-76.
 Icterus gravis neonatorum, 62.
 Impetigo, 50.
 Inanition, 59.
 Infant deaths, 11-12, 59-70, 160-1, 204-5, 209, 218-19.
 Infectious disease hospitals, 247.
 Infective disease, 60, 65-70, 73, 152-3, 222, 247.
 Influenza, 93-94.
 Institute of Social Medicine, 219.
 Interdepartmental Committee on the Care of Children (Curtis Committee), 16, 235.
 International Congress of Hygiene, 83.
 Intussusception, 76, 222, 259.
 Isaacson, V., 119, 129.
 Jackson, A. V., 89.
 Jackson, D. S., 48.
 Jackson, H., 215.
 Jacobsen, A. T. B., 114, 115, 129.
 Jakobsen, B., 114, 129.
 Janney, N. W., 119, 129.
 Jarrow, 202.
 Jaundice, 28-34, 36, 114.
 Jenner, E., 208.
 Jensen, C., 91, 109.
 Jewesbury, R. C., 128, 299.
 John and Temple Fellowship, 4, 128.
 Johns Hopkins Hospital, 6, 13.
 Kelleher, W. H., 101, 104.
 Kenny, Sister, 105.
 Keratomalacia, 49.
 Kerr, H., 6.
 Kling, C., 82, 83, 90, 109.
 King's College, Newcastle, 8.
 King's College, Newcastle, Department of Child Health, 13, 15-16, 22-23, 219.
 Knowelden, J., 88.
 Koplik, H., 206.
 Krehbiel, O., 120.
 Lactation, *see* Breast-feeding.
 Lactose, 115, 121, 124-5.
 Laevulose, 115.
 Landry, O., 82.
 Landsteiner, K., 81, 82, 83, 109.
 Lange, C. de, 74, 78.
 Lansing virus strain, 84.
 Laparotomy, 33, 64.
 Lartington Hall, 1, 3, 4.
 Laryngeal stridor, 63.
 Lawton, R. B., 99, 109.
 Learmonth, J., 14.
 Le Fleming, E. K., 300.
 Leslie, K. D., 4.
 Leslie, S., 13.
 Leukaemia, 283.
 Levinson, S. O., 87, 109.
 Lewin, P., 87.
 Lewis, P. A., 82, 108.
 Lewis, T., 234, 282.
 Lignac, G. O. L., 57, 58.
 Linacre Lecture, 18.
 Lister, W. A., 286.
 Liver, 7, 52, 55.
 Atrophy, 27-28.
 and Pernicious anaemia, 39-48.
 Liver extract, 39-48, 50.

- Liver tablets, 47.
 London University, 269.
 Lungs, 51-58; *see also* Pneumonia;
 Tuberculosis.
 Luton, 219.
 Lyle, R., 2.
- MacCallum, F. O., 101.
 McClosky, B. P., 88, 109.
 McCrudden, F. H., 119, 129.
 M'Donald, S., 33, 38.
 Macdonald, W. B., 74.
 McFarlan, A. M., 91, 109.
 MacGregor, A. R., 66, 70.
 MacLean, H., 115, 116, 118, 121, 122,
 129.
 McNeil, C., 10.
 Magee, H. E., 300.
 Malcolm, D. S., 96, 108.
 Malnutrition, 59, 142-57.
 Maltose, 115, 121.
 Mann, F. C., 38.
 Marasmus, 59.
 Marmion, D. E., 100, 109.
 Marmite, 72.
 Martin, J. K., 88, 109.
 Mastoiditis, 155.
 Maternal capacity, 174-203.
 and Breast-feeding, 161-73.
 and Medical care, 6, 181-2, 245-7.
 and Pink disease, 78.
 and Poliomyelitis, 93.
 Maternity clinics, 6.
 Maternity hospitals, 249-51.
Maternity in Great Britain, 219.
 Maxwell, D. M. W., 88, 109.
 Mead, R., 233.
Meadles, 50, 153, 155, 257, 288.
 Medical education, 221-4, 275-7; *see*
 also Nurses' education.
 Medical Officers of Health, 226-7.
 Medical research, 268-70, 281-92.
 Medical Research Council, 4, 7, 14,
 16, 19, 22-23, 219, 296.
 Report on the vitamins, 130.
 Medin, O., 81, 82, 109.
 Mehlhorn, L., 121, 129.
 Melnick, J. L., 90.
 Meningitic poliomyelitis, 97-98.
 Meningitis, 27-29, 52, 54, 66-68, 95,
 222, 239, 247.
 Mental abnormalities, 60, 61, 276.
 Mercury, 74, 78.
 Miller, F. J. W., 11, 15, 21, 59, 209,
 215, 217, 300, 302.
 Miller, J., 34, 38.
 Mills, S. D., 88.
 Milne, L. S., 33, 38.
 Milzer, A., 87.
 Ministry of Health, 173, 251, 298.
- Minor, G. R., 7, 39, 44-45, 48.
 Mogwitz, A. G., 120, 129.
 Montgomery, G. L., 67, 70.
 Moore, N., 233.
 Morant, R., 295.
 Moro, E., 72.
 Mothercraft, *see* Maternal capacity.
 Moyne, Lord, 8.
 Muir, G., 5.
 Mumps, 50.
 Murphy, W. P., 7, 39, 44-45, 48.
 Myalgia, 96.
 Myoclonic ataxia, 97.
 Myxoedema, 119.
- Naevus, 286.
 National Health Service, 14, 17.
 Neonatal sepsis, 59.
Neoplasm, 60.
 Nephritis, 62, 113-14, 117-19, 128,
 131, 165, 239, 257.
 Neuland, W., 51.
Neuritis, 76.
 Neuromuscular disorders, 62-65.
 Newcastle and Northern Counties
 Medical Society, 7, 19, 293.
 Newcastle Dispensary, 8, 143.
 Newcastle Regional Hospital Board,
 252, 253.
 Newcastle upon Tyne City Health
 Department, 6, 8, 142.
 Newcastle upon Tyne General Hos-
 pital, 11.
 Newman, G., 8, 59.
 Night-blindness, 49-50, 152.
 Nuffield Foundation, 219.
 Nuffield Provincial Hospitals Trust,
 Medical Advisory Committee, 13-
 14.
 Nurses' education, 277-9.
 Nursing, 245-7.
 Nutrition, 142-57; *see also* Breast-
 feeding.
 Nystagmus, 97.
- Officers' Training Corps, 2.
 Ogilvie, A. G., 12, 27, 66, 70, 143, 299.
 Onychia, 68.
Opisthoronus, 29.
 Opperman, F., 114, 129.
 Orthopaedic hospitals, 248.
 Osteitis, 68, 69, 222.
 Otitis, 68, 155.
- Paediatric consulting physicians,
 266-7.
 Palatal paralysis, 36.
 Paralysis, 80-109.
 Park, E., 206.
 Parkin, A., 40.

- Parsons, J. P., 118, 129.
 Parsons, L. G., 10, 80, 300, 301, 302.
 Paterson, D., 4, 10, 299.
 Patterson, D. H., 71, 79.
 Paul, J. R., 82, 83, 109.
 Peabody, F. W., 48.
 Pearson, C., 201.
 Peart, A. F. W., 285.
 Pellagra, 30, 152.
 Pemberton, R., 120, 129.
 Pemphigus neonatorum, 66.
 Penicillin, 242.
 Peptic ulcer, 30.
 Peritonitis, 27.
 Pernicious anaemia, 39-48, 162.
 Perrot, A., 63, 70.
 Pertussis, *see* Whooping-cough.
 Pettersson, A., 82, 83, 90.
 Photophobia, 31, 71, 75, 77.
 Pickering, G., 21.
 Pickles, W., 208.
 Picric acid, 118.
 Pink disease, 71-79.
 Pirquet, C. von, 206.
 Piszczek, E. A., 88, 108.
 Pleural effusion, 52.
 Pleurisy, 152.
 Pneumonia, 27, 50, 51, 66, 68, 76-77, 100, 152, 155, 239.
 Poliomyelitis, 80-109, 205, 257, 285.
 Popper, E., 82, 109.
 Population Investigation Committee, 219.
 Porphyrinuria, 61.
 Poynton, F. J., 299.
 Pregnancy, 87-89.
 Prematurity, 59.
 Princess Mary Maternity Hospital, 16, 163.
 Prolapse of the rectum, 76.
 Psychology, 271-80.
 Puberty, 192-3.
 Public Health Services, 219, 265-6; *see also* Child welfare clinics; Health visitors.
 Pyelocystitis, 68.
 Pyloric stenosis, 63-64, 165, 257.
 Pylorospasm, 62.
 Pyrexia, 35, 68.

 Quinsy, 247.

 Radiation, 50.
 Radiostol, 130-1.
 Respiratory disease, 214-15, 219, 257; *see also* Pneumonia; Tuberculosis; Whooping-cough.
 Reticulocytes, 40.
 Reticulocyte crisis, 44-47.
 Rheumatism, 288.
 Rhodes, A. J., 84, 89, 109, 285.
 Rice, J. L., 66, 70.
 Rich, A. R., 69, 70.
 Rickets, 50, 62, 125, 128, 130-41, 144-5, 149-50, 152-4, 207.
 Ridley, Lady U., 5, 12, 19, 21.
 Robertson, E. G., 89.
 Rockefeller Foundation Fellowship, 6.
 Rogers, D., 143.
 Rogers, J. C. T., 63, 70.
 Rolleston, H. D., 35, 38.
 Rolly, F., 114, 129.
 Romer, P. H., 81, 82, 109.
 Rowell, G., 5.
 Royal Army Medical Corps, 2.
 Royal College of Nursing, 278.
 Royal College of Physicians, 14, 233.
 Royal Navy, 2.
 Royal Society of Medicine, 286.
 Royal Victoria Infirmary, 4, 7, 11, 15, 16, 40.
 Rubella, 284.
 Russell, W. R., 87, 109.
 Rutherford, A., 34, 38.
 Ryle, J. A., 207, 219.
 Ryle, J. C., 96, 109.

 St. Bartholomew's Hospital, 233.
 St. Thomas's Hospital, 4.
 Sabin, A. B., 82, 89, 109.
 Saccharose, 121, 124-5.
 Sakaguchi, K., 114, 129.
 Salvarsan, 32, 35.
 Sanatoria, children's, 248, 258-9.
 Sandilands, J., 100, 109.
 Sanford, H. N., 165.
 Sargent, C. S., 119.
 Scarlet fever, 237, 239, 254-7.
 Scurvy, 50, 62, 144, 152-3.
 Seddon, H. J., 105, 106, 107, 109.
 Selter, P., 71, 78.
 Septicaemia, 67.
 Shattuck, L., 204, 206.
 Shaw, A. F. D., 6-7.
 Sheffield University, 269.
 Sickles, G. M., 84, 108.
 Silverberg, R. J., 89, 108.
 Simon, J., 206.
 Skin sepsis, 50, 68, 76, 153, 155.
 Smith, A. J., 8, 143.
 Smith, R. P., 299.
 Social medicine, *see* Field inquiries.
 Sodium chloride, 74, 78.
 Soule, E. H., 88.
 Spence, D. M., 1.
 Spence, J. C., 76, 79, 217.
 Birth and education, 1-2.
 Military service, 2-3.
 At Great Ormond Street, 4.

- Spence J. C. (*cont.*)
 Marriage, 4.
 Medical Registrar, Royal Victoria Infirmary, 5.
 Associated with Babies' Hospital, 5.
 Paediatric Physician, Newcastle General Hospital, 11.
 Honorary Physician, Royal Victoria Infirmary, 11.
 Nuffield Professor of Child Health, 15.
 Knighthood, 18.
 Death, 19.
- Spinks, A. F. G., 143.
 Splenomegaly, 53, 56.
 Squint, 253.
 Stabler, F., 163.
 Still, G. F., 104, 109.
 Strachey, L., 176.
 Strickland, B., 100, 109.
 Strouse, S., 117, 129.
 Students' Medical Society, 2.
 Sugar tolerance, 113-29.
 Survey technique, 110-12.
 Sussmilch, J. P., 206.
 Suzman, M. M., 48.
 Swedens, 90-91.
 Sweet, J. B., 79.
 Swift, H., 71, 79.
 Swift's disease, *see* Pink disease.
 Sydenham, T., 234.
 Syphilis, 32, 35, 66.
- Tachau, H., 114, 118, 129.
 Taylor, M., 12, 236, 251.
 Teaching hospitals, 263-70, 297-8.
 Tetany, 62.
 Theiler, M., 84, 109.
 Thompson, J., 62, 206.
 Thompson, R. B., 236.
 Thousand Families' Survey, 16, 209-29.
 Thrombosis, 44.
 Thursfield, H., 71, 79.
 Tonsillectomy, 30, 87-88, 237-8, 253-8.
 Tonsillitis, 95.
 Tracheotomy, 100.
 Trask, J. D., 83.
 Trotter, W., 175.
 Tuberculosis, 51-58, 64, 66, 69, 205, 238-9, 257.
- Tuberculous meningitis, 29, 52, 54, 239, 254.
 Turnbull, I., 1.
- Ungley, C. C., 48.
 University of Durham, 8, 15; *see also* King's College, Newcastle.
 University of Durham College of Medicine, 1, 7.
 University Grants Committee, 14, 16, 22, 268.
 University paediatric departments, 263-70.
 Uraemia, 35.
- Vaccination, 68.
 Van den Bergh test, 29.
 Venereal disease, 239, 257; *see also* Syphilis.
 Virus infections, *see* Infective disease.
 Vitamin A, 49-50.
 Vitamin D, 130-41.
 Vomiting, *see* Diarrhoea and vomiting.
- Walshe, F. M. R., 35, 38, 283.
 Walton, W. S., 217, 302.
 Ward, R., 90.
 Wardill, W., 5.
 Warkany, J., 74, 79.
 Wenner, H. A., 85, 90, 109.
 Wernstedt, W., 82, 83, 90.
 Wesselow, O. L. V. de, 115, 116, 118, 121, 129.
 West, C., 82, 98, 233-4, 249.
 Western Reserve University, 219.
 White, G., 177.
 Whooping-cough, 66, 213-14.
 Wickmann, J., 81, 82, 99, 109.
 Willcox, P. H., 88, 109.
 Willcox, W. H., 35, 38.
 Williams, H., 74, 79.
 Wilson, J. G., 164.
 Wilson's disease, 33.
 Wishart, M. B., 117, 129.
 Witts, L. J., 14.
 Wood, A. J., 73, 77, 79.
 Wood, L., 77, 79.
 Wood Committee, 60.
 World Health Organization, 18.
 Wright, E., 12.
- Xerophthalmia, 7, 49-50, 62, 144, 152-3.

